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Ophthalmology

Retinal Neovascularization

With Special Reference to Retrolental Fibroplasia

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Neovascularization of the retina commonly occurs in the following conditions: (1) Vasculitis Retinae; (2) Diabetic Retinopathy; (3) Von Hippel Lindau Disease; (4) Retrolental Fibroplasia. The first three conditions affect fully developed retinae. The last condition affects the premature retina. Although in each case the fundamental cause is different, the development and end result of these four conditions are very similar.

The theoretical conceptions presented in this article represent an attempt to supply an explanation for these changes. It is suggested that two separate mechanisms are involved.

(A) The Mechanism of Retinal Neovascularization.

When a vein becomes occluded a collateral circulation develops as a result of two processes. First, back pressure causes mechanical dilatation; and second, metabolites formed in the tissues distal to the blockage as a result of reduced blood supply cause capillary dilatation.

Ischaemia reduces the supply of glucose and other substances essential to the metabolism of living cells, but oxygen lack causes the most acute disturbance. This is particularly true of the cells of the cerebral cortex[1]. Since the retina is developmentally an outgrowth from the brain it may be assumed that the same holds true in retinal physiology .

Ballantyne and Michaelson[2] described various types of "new-formed" vessels which may be seen in the retina. New vessels may develop at the optic disc after thrombosis of the central retinal vein. After thrombosis of a vein at an arteriovenous crossing, new vessels may develop in an attempt to circumvent the obstruction. They considered that venous obstruction and the local production of metabolites are the immediate causes of vasodilatation and retinal neovascularization.

(1) Vasculitis Retinae.

The etiology of this condition is not understood, but most authorities consider that tuberculosis plays an important part. A patchy inflammation affects segments of the retinal vessels, particularly venules at the periphery. Exudates and hemorrhages appear adjacent to the affected segments.

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Some cases do not progress beyond this stage, but others develop tufts of new vessels in relation to the patches of vasculitis. Ballantyne and Michaelson[2] state that the entire thickness of the vessel wall may be involved, leading to partial or complete occlusion of the lumen. Local ischaemia would appear to be the immediate cause of neovascularization.

(2) Diabetic Retinopathy.

In this condition the chief pathological changes are the development of micro-aneurysms in the venous side of the capillary network with dilatation and tortuosity of the veins.

Both Ashton[3] and Ballantyne[4] consider that venous stasis is an important factor in development of the condition. Thrombosis of the central retinal vein and its branches are common. New vessel formation occurs late in the development of diabetic retinopathy, and it is suggested that by the time this state is reached venous stasis is severe. Again it would appear that a relative ischaemia is the immediate cause of neovascularization.

(3) Von Hippel Lindau Disease.

This disease shows dominant inheritance with angiomata and cysts occurring in the cerebellum, medulla, pancreas, kidneys, and adrenals. In the eye the usual picture is that of an artery and vein joining to form a small tumour. This tumour enlarges and the artery and vein become dilated and tortuous so that it is difficult to distinguish one from the other.

The tumour is composed of angiomatous capillaries and may form an arteriovenous aneurysm. This must cause inadequate blood and oxygen supply to the capillaries of adjacent retina. Exudates occur and some cases develop new vessels on the peripheral side of the angiomatous tumour. Whilst this condition is fundamentally neoplastic, it is suggested that these new vessels grow in response to metabolites caused by locally diminished blood supply.

(B) The Mechanism of Development of Retinal Detachment.

In vasculitis retinae, diabetic retinopathy, and Von Hippel Lindau disease, new vessels may develop in the retina, and it is these new vessels which rupture and give rise to haemorrhages. Complete retinal detachment with secondary iridocylitis and secondary glaucoma may be the end result in all of these conditions.

Small haemorrhages may remain within the retina, but large haemorrhages burst through the

internal limiting membrane into the vitreous. Sometimes a haemorrhage absorbs rapidly, but more often a vitreous haze persists and repeated haemorrhages occur. It is in the type of case in which blood persists in the vitreous that new blood vessels may grow from the retina into the vitreous itself. The new blood vessels or rete mirabile may arise from the optic disc, or less frequently from elsewhere in the retina.

Wolff[5] states that the rete mirabile arises from the optic disc because in this situation there is no internal limiting membrane. In other situations a rete mirabile occurs only if the internal limiting membrane is ruptured by a haemorrhage so that the retina is in direct contact with the blood. He considers that the growth of new vessels into the vitreous is the result of a similar mechanism to that stimulating the growth of new vessels into a haematoma. A fibroblastic reaction and organization lead to the umbrella shaped retinal detachment which may occur in each of these three conditions.

Experimental and Clinical Observations in Retrolental Fibroplasia.

The developing retina is supplied by the hyaloid system which reaches its maximum development about the end of the third month. Thereafter the hyaloid system regresses and the retinal arterial system begins to develop. It buds out from the hyaloid artery in the region of the optic disc and grows peripherally, reaching the ora serrata at eight months (Mann[6]). In premature infants, therefore, these peripheral budding terminations of the retinal vasculature may still be in a state of rapid development at birth.

The retinal vessels of kittens are incompletely developed at birth and reach the ora serrata three weeks after birth. They, therefore, resemble those of the human premature infant. Ashton[7] exposed newborn kittens to high concentrations of oxygen, viz., 60-80% for several days. He found that developing retinal vessels were obliterated, and that the degree of obliteration was proportional to the immaturity of the retinal vessels, the concentration of the oxygen and to the duration of the exposure. On transferring the kittens to a normal atmosphere or an oxygen tension of 20%, new vessels developed from the retina and haemorrhages occurred.

Patz, Eastham, et al.[8] reported that rearing newborn rats and mice in 80% oxygen caused retinal vasoconstriction and changes resembling retrolental fibroplasia, especially if the exposure was prolonged. Transfer to air did not appear to be necessary to cause these changes.

Szewczyk[9] observed that a state of hypoxia or relative anoxia in a premature infant, whether at birth or during the first few weeks after birth, caused oedema and vasodilatation of the retina, a condition which he called hypoxic retinopathy. Anaemia or upper respiratory infections may cause an inadequate oxygen supply and produce the same retinal appearances. He found that hypoxic retinopathy did not occur if the oxygen tension was reduced slowly, and that in the early stages the condition regressed if the infant was returned to the increased oxygen tension. He recommended that oxygen should be given to premature infants only when cyanosis demands it. Jefferson[10] has also reported the regression of retrolental fibroplasia on treatment with high concentrations of oxygen. Crosse and Evans[11] considered that premature infants who were nursed in incubators with high oxygen tension became acclimatized to the increased oxygen, and that on removal from the incubator the sudden drop in oxygen tension caused the disease. Szewczyk also holds this view.

Reese[12] has reported finding extramedullary foci of haemopoiesis in the ureal tract of the eyes of cases of retrolental fibroplasia. Extramedullary foci of haemopoiesis are not uncommon in premature infants[13], but this finding may support the theory that ischaemia is the underlying cause of this condition.

Mechanism in the Development of Retrolental Fibroplasia.

It would appear that high oxygen tension causes vasoconstriction of the retinal vessels. If high oxygen tension is prolonged, occlusion of the vessels occurs and leads to ischaemia. Some degree of adjustment appears to be possible, but a sudden reduction in oxygen tension adds a relative anoxia to the relative ischaemia and aggravates the condition.

The retinal vessels in a premature infant are in a state of rapid development at the periphery. Relative ischaemia would therefore affect the retinal vasculature more severely peripherally than centrally, and it is an observed fact that the condition begins peripherally.

Reese[12] has described in detail the development of new vessels resembling angioblastic formations in the nerve fibre layer of the retina. That the almost embryonic retinal vessels when stimulated to new vessel formation should resemble an angiomatous tumour is to be expected. The new blood vessels show exuberant growth and break through the internal limiting membrane to invade the vitreous.

Haemorrhages occur into the vitreous so that the vitreous resembles a haematoma. Organization of the vitreous haemorrhage causes the end stage of retrolental fibroplasia.

Summary

(1) Although the causes of vasculitis retinae, diabetic retinopathy, Von Hippel Lindau disease, and retrolental fibroplasia are different, it is suggested that similar factors operate to produce similar end results.

(2) In each it appears that reduced blood supply or relative anoxia lead to vasodilatation and neovascularization in the retina.

(3) Haemorrhages from these newly formed vessels rupture into the vitreous so that it resembles a haematoma. New blood vessels grow into the vitreous as into a haematoma, and the resulting organization causes the retinal detachment which is common to all four conditions.

(4) It is suggested that in the premature infant high oxygen tension causes constriction or occlusion of the retinal vessels. Ischaemia occurs and is aggravated by the relative anoxia produced by the change to normal oxygen tension. This stimulates the almost embryonic retinal vessels to exuberant neovascularization, which leads to the chain of events we recognize as retrolental fibroplasia.

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Othopedics

Tuberculosis of the Greater Trochanter

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1. In Indians.

A recent article on the subject of tuberculosis of the great trochanter states that relatively few cases have been recorded. Meyerding and Mroz in 1933 analysed a series of 19 cases from the Mayo Clinic. In the discussion that followed the presentation of their paper, it was evident that few orthopaedic surgeons had seen more than the occasional case. In 1921, Peabody described 3 cases involving the great trochanter out of a total of 3,062 lesions of bone, tuberculous in character. In 1942 Donovan and Merrill gave particulars of 5 cases while in 1947, McMurray reported on 12 cases collected over a period of 15 years. The incidence of cases affecting the great trochanter in bone tuberculosis was estimated at 2 per 1,000. It may therefore be of interest to report on the findings at Brandon Sanatorium. All the patients admitted to this institution are Indians or Eskimos. Since the opening of the Sanatorium in June 1947, 860 cases have been admitted. A few after investigation have been found to be non-tuberculous, but even taking this into account, no fewer than 6 cases of trochanteric tuberculosis have been found. There were 82 cases of tuberculosis of bone and joint, and of these the 6 cases affecting the great trochanter amounted to 7.31%, a figure many times higher than any previously quoted. It would probably not be fair to regard this proportion as typical of the state of affairs among the Indian population. Most likely it is accounted for by more thorough efforts to promote the welfare of the Indian than have been customary in the past. Be that as it may the number of cases met with appears to be quite exceptional.

Of the 6 cases, 5 were male and 1 female, the latter an Eskimo from Coral Harbour, Southampton Island. The youngest was 10, and the oldest was 43. Occupation was not a factor. One out of the 6 gave a history of previous trauma; in 1943 some logs fell on his right hip and thigh producing an open sore which did not heal for 18 months and which had broken down again. Rather striking was the interval between onset and admission to hospital. In 4 cases it was 13 years, 10 years, 29 years, and 7 years respectively. In the case of the Eskimo girl information on this point was not available because of difficulties of language. In the case of the lad of 10, the swelling was noted 9 months before admission, and the "abscess" had been incised 3 months before arrival in hospital.

Though no definite information was obtained regarding contact with tuberculous subjects, it is practically certain that all of the patients had been exposed to infection. Tuberculosis elsewhere in the body was noted in 5 of the 6 cases. In 4 of these the lungs were involved; one (aged 10) moderately severe, the others mild or arrested. Skin, lymphatic glands and genito-urinary system were not obviously affected in any. Two showed disease of the spine, spontaneously arrested, and 2 showed a focus in the ischium.

As regards symptoms, pain was notably absent; only one complained of soreness. One had a limp

with some restriction of movement at the hip-joint. In this case there was a focus of infection in the ischium. Two showed swelling in the neighborhood of the great trochanter, and 5 showed the presence of sinuses, one, two, or three. The general health of all was good, except in the case of the boy aged 10 with active lung infection.

In none of the others was there any debility or muscle wasting. Temperature was never elevated more than a degree or so above normal. The sedimentation rate, (Westergren) was generally raised, the average being 50, the lowest 18, the highest 92. In all cases the Wassermann reaction was negative.

Radiological findings were almost uniform, showing erosion of the lateral surface of the great trochanter, and in most cases irregularity of the bone structure of this process. Donovan and Sosman sound a warning that too heavy exposure may obscure the changes in the trochanter.

Differential diagnosis would not seem to be difficult, although one case, (Keith) was mistaken for sarcoma, and Kidner speaks of the condition being mistaken for fibro-cystic disease of bone. There does not appear to be grave risk of extention of the condition to the hip-joint.

The pathological sequence is not entirely clear. It is usually assumed that the primary focus is in bone and is of the nature of an epiphysitis or metaphysitis. According to this view the bursa underlying the gluteus maximus is involved secondarily. In one of our cases, the "pus"-containing cavity extended from the outer aspect of the great trochanter almost as low as the knee joint. In another case, of which the operative record is given below, the gluteal bursa did not seem to be involved. In only one of our cases was a definite sequestrum found, although cavitation of the bone was found in 3 cases. The question "Which comes first, the bursa or the bone?" would appear to be of academic interest only; it may be safely assumed that both require attention in every case. In the case quoted below, the detailed findings strongly suggest that in this patient at least, the primary lesion was in bone.

Treatment involved open operation in 5 cases. The lad of 10 healed completely without the use of streptomycin or other medication. A typical operative record is as follows.

"Fascia lata was incised. Yellowish material was seen to be bulging through the attachment of gluteus medius to outer side of the great trochanter. Gluteus medius was cut free from its attachment and immediately a mass that looked almost like a bursa with thickened walls was disclosed. It was possible to dissect all round this and remove it as one mass. It communicated with a small cavity in the trochanter and this cavity extended to the deep surface of the trochanter. A small white sequestrum was found lying in the mouth of this cavity. The cavity itself was thoroughly curetted. The wound was sutured without drainage. No plaster".

After-Treatment

One patient had a plaster spica applied. The others were sutured with or without a 24-hour drain, and all healed per primam. All had Penicillin for 48 hours, and Streptomycin ½ gm. daily for about 6 weeks. Subsequent history shows that one case, the only one treated with a spica, broke down after 5 months, but healed again within 2 months. A second case had about 60 cc. of straw-coloured fluid aspirated from a swelling 48 days after operation. Culture of this fluid showed no tubercle bacilli and the wound did not break down.

In four of the cases histological report on the tissue removed confirmed the diagnosis of tuberculosis. One case did not undergo operation, and in another case, unfortunately, the tissue was not submitted to microscopic examination.

As a result of the unusual experience related above, it was decided to make enquiries from all of the Indian hospitals of Western Canada. This was carried out by Dr. W. J. Wood, and replies were received from the superintendents of the Indian Hospitals at Fort William and Moose Factory in Ontario, Prince Albert in Saskatchewan, Edmonton in Alberta, and from Sardis, Nanaimo, and Prince Rupert in British Columbia. Only one case of trochanteric tuberculosis was discovered. That was at Edmonton.

"We have only one case in our records of tuberculosis of the great trochanter and she is in the hospital at present for treatment. She is C. M., age 17, an Indian girl from Fort Wrigley. She was admitted here June 19th, 1951, with Pott's Disease of lumbar three and four and also complained of pain over the right great trochanter. Pus was aspirated from the subgluteal bursa which was positive for A.F.B. on culture and guinea pig. X-ray revealed slight erosion of tip of trochanter but hip-joint itself showed no bony change.

We have between 60 to 70 patients here with bone and joint tuberculosis and a total of 440 with tuberculosis of some form or other. There are another approximate 300 under treatment in the Mission Hospitals of the North West Territories, but do not know of any of the latter patients that have tuberculosis of the greater trochanter".

W. F. L.,
Medical Superintendent.

11. In Whites.

The discovery of no fewer than 6 cases of tuberculosis of the great trochanter within the space of a few months and within a single sanatorium, naturally prompted an enquiry as to the frequency of this condition amongst non-Indians. Requests for information were directed to Mani-

toba Sanatorium at Ninette, to St. Boniface Hospital and Sanatorium and to the General Hospital, Winnipeg. Private records were also searched. Summaries of seven are appended

1. F. A., aged 19, nurse in training at Misericordia Hospital, was admitted with a swelling over the right great trochanter. X-rays showed "no abnormality in the bodies of the lumbar vertebrae and in their intervertebral discs. Pelvic bones normal. Cystic process in the greater trochanter of the right femur". On 18th April, 1929, the right great trochanter was opened up, tissue removed, and sent for microscopic examination. The findings were:

The soft tissue consists of a mass of fibrin infiltrated with inflammatory round cells.

Fluid contents of cyst, right hip.

No Tubercle Bacilli found on staining.

No growth on culture.

Comment: After the lapse of more than 20 years the written report of this case is quite inadequate to establish its claim, and the subsequent history is not obtainable. The impression on the mind of the surgeon at the time of operation was that the condition was tuberculous in character, and that impression has been strengthened rather than weakened by further experience.

2. Mrs. W. K. C., aged 40, housewife, formerly nurse.

20-10-39. Complained of pain about the left hip of many years duration. In 1934 had tonsils removed. No improvement. Examination showed some limitation of movement in all directions at left hip-joint; tender nodules, left buttock. X-ray showed roughening of the outer side of the left great trochanter.

21-10-39. Operation.

The left great trochanter was exposed. Over it there was a bursa about the size of a crabapple, partially broken down in the centre. This communicated with a gutter in the great trochanter. The bone about the edges of the gutter was rough. The bursa was dissected away, the rough edges were smoothed, and the soft parts closed.

26-10-39. Microscopic report of tissue, "Tuberculosis".

23-2-45. Returned complaining of some pain about the old scar. Some swelling and tenderness over the left great trochanter. X-ray showed proliferation of periosteum on outer side of trochanter.

29-1-46. Condition not much changed since last visit. The whole mass was excised.

29-1-46. Report on tissue "probably tuberculosis".

August, 1951. Report from a neighbour that patient is doing well.

3. Mrs. R. H., aged 27, Housewife, admitted to St. Boniface Hospital 26-4-48.

Complained of discharging sinus left thigh. About 2 months before admission an almost painless swelling appeared over the left greater trochanter. Four weeks later it broke down and a persistent discharging sinus had been present ever since. Penicillin therapy from April 26th until 5th May, 1948.

On 22nd May, 1951, the sinus was excised and the greater trochanter at the origin of the sinus was curetted. On 31-5-51, it was reported that a positive culture of Tubercle had been obtained after 4 weeks incubation of material from the sinus.

4. J. D., aged 64, labourer, Galician.

Came to Winnipeg General Hospital, 30-11-48. He complained of pain in lower back and right leg for about 8 years. He came to Canada 1918. Speaks no English. Always a general labourer. Cough for many years. Last 8 months on Social Aid, $28 per month. Food often insufficient. He was pale; in no apparent distress; atrophy of right quadriceps; pain on rotation of right thigh. X-ray showed area of destruction right great trochanter. Condition considered probably tuberculous. Treatment refused.

On 7-11-49 he was admitted to Winnipeg General Hospital. Over the right buttock there was a broken down area red, indurated, weeping. Similar area right scrotum. Mantoux negative for 1/10,000. Sputum positive for Tubercle. Sedimentation rate 30. Hm. 89%, W.R. negative. X-ray showed practically no change in condition of right great trochanter from that reported on a year previously. The left hip-joint showed marked disintegration. Condition was regarded as too poor to permit of operation. Patient was sent to King Edward Hospital.

5. (The next two reports I owe to the kindness of Dr. A. L. Paine).

"Miss E. S. Has been on treatment for far advanced bilateral pulmonary tuberculosis since March 1949. Orthopaedic history goes back to August 1945 when she was examined at the Central Clinic because of a swelling over the left great trochanter. This was considered to be a tuberculous bursitis and cleared up after several aspirations. At that time tubercle bacilli were not found.

Since coming on the cure her hip has been bothering her again, mainly in the matter of swelling and pain when lying on it and we have aspirated fluid from it on several occasions, namely July 1946, February 1951, July 1951, and October 1951. Fluid has always been clear and green, negative for tubercle bacilli on smear but positive to culture. Also a guinea-pig injected with fluid in February 1951 was positive for tuberculosis on May 4, 1951.

X-rays have not been considered definite for bony involvement and we are planning to dissect

out this soft tissue area containing the fluid.

Her pulmonary tuberculosis has improved markedly. She had a left-sided thoracoplasty and a pneumothorax on the right side and has taken several courses of streptomycin. Sputum is now negative for tubercle bacilli".

6. Mrs. N. W., aged 69. Admitted to Ninette March 1951, complaining of painful hip since November 1950. Previous treatment included the application of heat and incision of a swelling. From this swelling tubercle bacilli were isolated. Over the left thigh there was a diffuse swelling with a rather indolent looking sinus lined with grey necrotic tissue and with undermined edges. Pus from the sinus was negative for tubercle bacilli but positive to culture. She was treated with streptomycin and PAS and the sinus closed. There was a persistently high sedimentation rate, (87 mm Westergren). She was transferred to St. Boniface General Hospital on 26th October, 1951, and was operated on. The left greater trochanter region was explored, the bursa overlying this prominence was enlarged and filled with fairly thick pus. This was excised and the underlying bone appeared to be normal. The lateral cortex was excised and the medulla found to show small pockets containing yellow pus. It was necessary to excise practically all of the greater trochanter, and the diseased medullary bone was curetted down to normal bone. This was carried well up into the neck of the femur. Post-operatively she had extension and penicillin.

7. Particulars of a seventh case have been kindly supplied by Dr. Tubber Kobrinsky.

Mrs. F. C., aged 59, housewife (from Alberta).

20-7-49. Reported complaining of pain, swelling, limitation of movement at the left hip. X-ray showed irregularity of the great trochanter, with calcification in the soft tissues over this area.

28-7-49. Exploration of the affected region was carried out. A large irregular thin-walled cystic mass was found attached to the bone from the level of the great trochanter to junction of upper and middle thirds of the femur. The cyst was filled with purulent cheesy material. Although the condition was judged clinically to be tuberculosis, examination at the pathological laboratories of two institutions were both indecisive. Anti-biotics were prescribed, and patient left for home.

3-11-52. At this visit patient still complained of pain, and an X-ray showed irregular changes in the substance of the great trochanter. Biopsy was again carried out. On this occasion examination of the swabs from the hip, elicited a positive diagnosis of Tubercle, as did culture and at a later date the results of guinea-pig inoculation.

The patient was transferred to Calgary Sanatorium. On 29-1-53 the left great trochanter was resected and the tuberculous tracts excised. A plaster spica was applied, and the patient was immobilised for 3 months. Weight-bearing was allowed gradually after this.

Summary

Particulars are given of 14 cases diagnosed as tuberculosis of the great trochanter. Of these the majority were confirmed by laboratory investigation. Seven of these cases occurred in Indians and seven in white subjects.

Conclusions

1. Tuberculosis of the great trochanter is a relatively uncommon condition. It tends to be chronic existing for months or years before being recognized and treated.

2. Pain is not a prominent symptom; the general condition of the patient may remain comparatively satisfactory.

3. Any lesion affecting the cortex of the great trochanter should be presumed to be tuberculous even in the absence of confirmation by microscopic examination of tissue, culture of "pus", or guinea-pig inoculation.

4. Incomplete removal or conservative treatment is apt to be followed by recrudescence of symptoms.

5. Streptomycin and similar anti-biotics may be of benefit.

6. Complete excision of the area involved, including thorough treatment of the bone lesion, usually leads to complete recovery.

Medicine

Prognosis in Poliomyelitis

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The history of poliomyelitis falls quite clearly into three phases. The first starts at a remote period in history and is indicated by paleopathological evidence and by some vague references in classical literature. Whether Samuel[1], when he writes of the lameness of Mephibosheth was referring to poliomyelitis (as suggested by Sir Wm. Osler in his textbooks) and whether Homer[2] visualized a similar cripple when he describes Vulcan's disability is a matter for diverting but unprofitable speculation.

The second period begins with the first good clinical account by Underwood[3] in 1789. During the following century most textbooks described the condition and thirty references are listed by the National Foundation[4]. During this period the disease was comparatively rare, truly infantile, entirely sporadic, mostly confined to the lumbar cord and rarely fatal.

The third phase began about 1880, since when all of the earlier features have gradually changed. It has become a common epidemic disease, now being, after influenza, the most serious infectious disease in civilized communities; it is no longer confined to infants, recent epidemic showing as high an incidence in those over twenty as among those under five; cervical cord, bulbar and cerebral involvement have become more frequent and deaths have become common. Because of this rapid and continuing change in all important features it is difficult with confidence to predict the future.

A great variety of opinions have been expressed with regard to prognosis. Some observers dwell on the great liability to crippling and others point to the undoubted tendency to spontaneous recovery. In various epidemics and under different circumstances the death rate is stated to be from two to twenty per cent, the paralysis incidence from ten to seventy-five per cent and the residual paralysis from ten to eighty per cent. These very divergent findings depend mostly upon changing criteria for diagnosis and also on the changing character of the disease. Until about thirty years ago the diagnosis was rarely made except in paralysis cases. Now it is commonly made on very slight symptoms and there is no doubt that in some epidemics every suspected case has been recorded and in others only those that are paralyzed find their way into official records. Many cases undoubtedly due to other viruses (especially Coxsackie) have been in recent epidemics counted as poliomyelitis. Official notification by practitioners and recording by Health Departments have also been irregular; the true situation as to morbidity and mortality is therefore impossible to estimate.

Present evidence suggests:

1. In any epidemic a large proportion of susceptibles become infected.

2. Of those infected only a small number have any symptoms.

3. Of these, again, only a few are clinically ill and are recorded by Health Departments.

4. Further, only about half of these develop paralysis.

5. Of these who develop paralysis the vast majority recover without a crippling disability.

6. The total death rate is very small, usually being about 5% of those notified.

In its ultimate analysis therefore, infection by these viruses is relatively innocuous.

To predict the fate of those cases that survive, but have paralysis, should be much less difficult. But here also there is an enormous variety of opinions. Sister Kenny's extraordinary contention that under "orthodox" treatment only 13% of patients recover without paralysis is of course too fantastic to be given credence by anyone who has witnessed the enormous spontaneous recovery that can take place with no specific treatment whatever. Some observers have made an effort to express precisely what degree of recovery may be expected in various degrees of paralysis.

Harry[6] in 1938 reported the final results on fifty cases followed for two years. His conclusion may be summarized as follows:

Slightly paralyzed muscles (i.e. those which contract against gravity and resistance) "became more powerful in six months and many were eventually regarded as normal."

Severely paralyzed muscles ("showing only a flicker") showed improvement in only 10%, these "became powerful enough to contract against gravity." Immediate degrees of paralysis showed corresponding improvement.

R. L. Bennett[7] has made an effort to estimate the percentage of improvement from month to month. He believes that sixty per cent of the total ultimate recovery occurs in the first three months and twenty per cent in the second three months, when it is completed.

Watkins[8] in a paper entitled "Progressive Disabilities in Poliomyelitis," attempts to show the rate of recovery in various grades of paralysis. He indicates that completely paralyzed muscles show very little improvement at the end of a year; in those with slight paralysis fifty per cent

are normal in six months and ninety per cent are normal in one year. Intermediate degrees of paralysis (graded as "trace," "fair minus," "fair," and "fair plus") show corresponding degrees of improvement after six and twelve months.

These and other observers have each used a different method for expressing the strength (or weakness) of a muscle. None of the methods lead themselves to statistical analysis, nor can one be compared to the other.

It is unfortunate that more precise progress reports are not available. This deficiency in the literature gives rise to the great divergence of opinion as to prognosis. It also accounts for the fact that various methods of treatment cannot be compared. Devotees of each "system" claim good results but rarely give convincing data. Since most cases are now followed by trained physiotherapists who express degree of paralysis or strength numerically and not by symbols or vague terms, it should be possible for various observers to produce comparable figures. Such comparisons would be facilitated if some international method of recording were adopted. A common practice in Britain is to record the degree of muscle power by the figures 5, 4, 3, 2, 1, 0 (5 being normal strength—0 complete paralysis). Another common method is to record the amount of paralysis, 5 indicating complete paralysis and 0 meaning normal muscle. Either of these methods is suitable and one may be readily changed to the other by inversion.

This study deals with the course of thirty-six moderately or severely paralyzed patients carefully observed for eight months. These were selected from eighty-six patients with paralysis who were examined on several occasions. Excluded from the study were those with cranial nerve and bulbar involvement only, those with very mild paralysis, those who could not be accurately assessed (under two years of age), those who could not be closely followed and two cases that have quadraplegia and require constant care in respirators. The residual group of thirty-six was composed of nineteen male and seventeen female patients. The age distribution was similar to that of the total epidemic (840 cases throughout Manitoba) and is shown in Chart 1.

Chart 1

	0-4	5-9	10-14	15-19	20+
Female	4	5	5	1	2
Male	7	6	3	0	3

Method of Treatment

Seven of the thirty-six under study were treated during the acute stage (from two to six weeks) in a fully equipped Children's Hospital, where they received a modified Kenny Regimen. All other treatment during the eight months after onset was given in a small rural hospital of thirty beds (Winkler, Manitoba). All were hospitalized for a time but most of the treatment was conducted in the home or by visits to the hospital. The work was supervised by the three local doctors and all the physiotherapy was done by one physiotherapist who had had long experience with poliomyelitis and peripheral nerve injuries. She was assisted by one enthusiastic but untrained volunteer.

Rehabilitation in the home was encouraged at the earliest possible moment; the longer this is postponed the more difficult it becomes. As soon as the hospitalized patient returned home responsible members of the family were shown how to give passive movements and re-education where indicated. Schemes of exercises and their progressions throughout the various stages were carefully taught. The fullest possible co-operation was enlisted from the patients' relations and they accepted responsibility and responded well, taking great pride in the patients' progress.

For school children mildly affected, group exercises were organized and proved very popular.

During the acute stage (i.e. while constitutional symptoms, much pain or tenderness were still present) no passive stretching to muscles in spasm was given and complete rest in the optimum position was encouraged. As soon as the patient began to show signs of activity very gentle passive movements, within the limit of pain, and active re-education were begun. No heat in any form, no massage, electrical treatment or tub baths were used. Except in the first few cases no lumbar puncture was done. Splintage was reduced to a minimum.

In the later stage and as the weather became colder, radiant heat was applied to out-patients whose paralyzed limbs showed evidence of cyanosis. No massage or electrical treatments were employed at any stage, physiotherapy consisting entirely of detailed re-education to paralyzed muscles and passive movement to those joints unable to perform their full range of movement.

A room was equipped with wall-bars, fixed cycle, sling circuits, abduction pulley, long mirror, invalid walker and a variety of baby walkers. Similar equipment was installed in the home when indicated.

Method of "Scoring"

Each patient was fully assessed as to muscle paralysis on several occasions during the eight months of observation. This consisted of expressing the amount of paralysis found in each muscle group by a figure, from 5 to 0. The progress of each muscle group could thus be followed from month to month. This was estimated for each limb, the neck muscles being included with the upper limbs, and the abdominal and back muscles with the lower.

The figures allotted for various degrees of paralysis were as follows:

No discoverable contraction	Grade 5
A "flicker"	Grade 4
Acting but not against gravity	Grade 3
Acting against gravity	Grade 2
Acting against gravity and resistance	Grade 1
Normal	Grade 0

These figures throughout will be referred to as "grades" 5, 4, 3, etc. The totals for any limb or any patient will be called "The score."

Every muscle was not separately scored; in some cases their importance did not seem to justify it (e.g. the hand muscles) and in some cases function could not be differentiated (e.g. infraspinatus and teres minor). In the upper limb 23 and in the lower 27 muscle groups were followed in each case. These are listed below—100 in all, giving a total possible score of 500.

UPPER LIMB

Neck—
Flexion.
Extension.

Shoulder Girdle—
Trapezius
Supraspinatus
Pectoralis Major.
{Latissimus Dorsi
{Teres Major.
Serratus Magnus
Rhomboids.

Arm—
Deltoid.
Biceps
Triceps.
Brachialis Anticus.

Forearm—
Supinator Longus.
Extensor Carpi Radialis (Long and Brev.)
Extensor Communis Digitorum.
Extensor Carpi Ulnaris.
Extensor Pollicis Longus.
Extensor Pollicis Brevis.
Flexor Carpi Radialis.
Flexor Sublimus Digitorum
Flexor Profundus Digitorum.
Flexor Pollicis Longus.

Hand—
Thenar muscles.
Hypothenar muscles.
{Interossei
{Lumbicales.

LOWER LIMB

Back—
Erector Spinae.
Quadratus lumborum.

Abdomen—
Rectus Abdominis.
Oblique and transverse abdominal muscles.

Thigh—
Ilio-Psoas.
Gluteus Maximus.
Gluteus Medius.
Tensor Fascia Femoris.
Internal Rotators.
External Rotators.
Quadriceps.
Hamstrings — inner.
Hamstrings — outer.
Adductors.

Leg and Foot—
Tibialis Anticus.
Extensor Longus Digitorum.
Extensor Longus Hallucis.
Peronei.
Gastrocnemius and Soleus.
Tibialis Posticus.
Flexor Longus Digitorum.
Foot Intrinsics.

It will be seen that a totally paralyzed upper limb (including the neck) would have a "score" of 27 x 5 or 135 and a totally paralyzed lower limb (including back and abdomen) would have a score of 23 x 5 or 115.

Chart 1

Chart 1 shows the progress in each limb of a moderately severe case from the second to the eight month. This six-year-old girl had spent the first five weeks in the Children's Hospital (Winnipeg) and was seen by us in her fifth week. It will be seen that she then had involvement of all four limbs, her total score being 114, which is the equivalent of 23 totally paralyzed muscles. As a matter of fact she fortunately had no total paralysis but a very widespread less severe involvement. There was no muscle graded "5" and only one was graded "4"; all the others involved (a total of 62 out of a possible of 100) were in the 3, 2, or 1 groups. For that reason she made dramatic improvement and after eight months has a score of only 20. The residuum is due to persistent weakness (grade 3) in the abdomen, right iliopsoas and right adductors. All other groups at the end of observation approached normal strength.

Chart 1

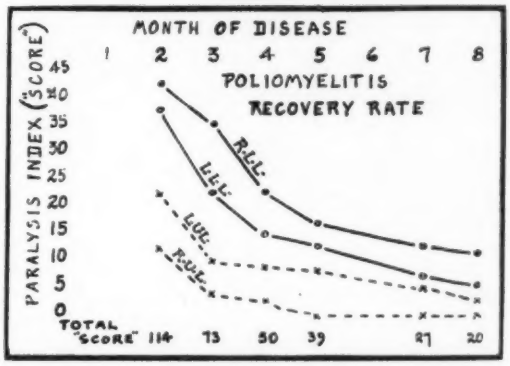


Chart 2

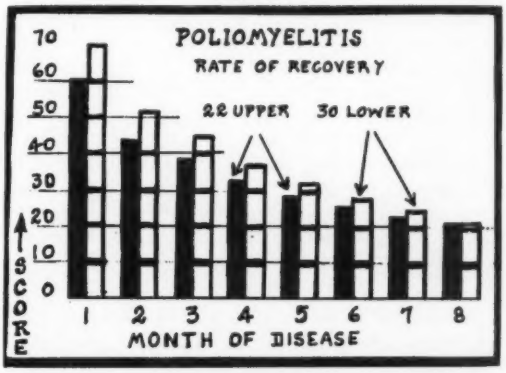
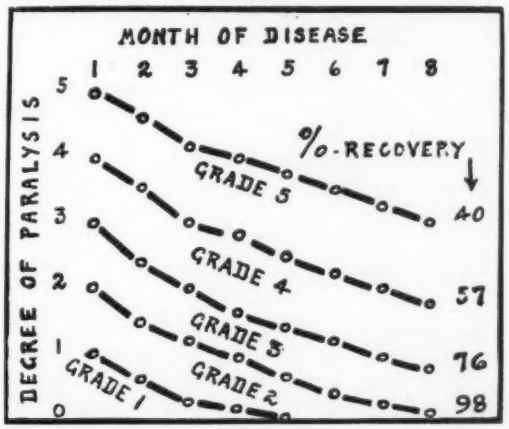


Chart 3



The chart shows, what has often been demonstrated, that is, very rapid improvement during the first four or five months and less rapid recovery thereafter.

In order to show the rate of recovery in the whole group the average monthly scores for all the upper limbs and all the lower limbs are shown in Chart 2. This again shows rapid progress during the first five months (65 to 30) and relatively less thereafter (from average of 30 to 20).

Chart 2

The percentage reduction in the original paralysis of the whole group in each month and the rate of improvement is shown in Table 1.

Table 1

Percentage of Original Paralysis Month by Month

Month.	1st	2nd	3rd	4th	5th	6th	7th	8th
% of original	100	73	65	54	47	40	37	32
Rate by month ..		27	8	11	7	7	3	5

The 27% improvement in the first month is not all due to recovery from paralysis. Though the first assessment was not made during the acute period, there are always other elements that contribute to a defect in voluntary movement at the first examination. Such factors are, muscle tenderness, hyperaesthesia, cerebral irritability and consequent indifference or antagonism. Much of this disappears after the patient has had a short course of treatment, has learned something of the technique and decided to co-operate; then a truer estimation of actual muscle weakness becomes possible. After the second month improvement continued at about the same rate (7 to 11%) till the seventh or eighth month, when it became less. This residuum was almost entirely due to persistence of weakness in a few muscles originally graded as "5."

Chart 3

The rate of improvement in the five grades of paralysis was computed and is shown in Chart 3. The total percentage improvement in each grade from 5 to 1 was respectively, 40%, 57%, 76%, 98% to 100%. In fact all those that were graded "1" had completely recovered within five months. Of those marked as "2" (113 muscles) all but twelve had made a complete recovery in eight months. Recovery in the other grades was not complete. Table 2 tabulates the final result in each grade of paralysis. The improvement in six hundred and eighty-eight muscles is shown in percentages.

Table 2

Original Examination		Final Examination: % in each grade					
Grade	No. of Muscles	5	4	3	2	1	0
5	128	18	21	35	7	10	9
4	57		11	19	35	21	14
3	165			1	20	30	49
2	113					10	90
1	225						100

This table shows that only 9% of muscles originally considered to be completely paralyzed recovered entirely and that 74% remained in Grades 5, 4 or 3. In contrast 100% of Grade 1 paralysis recovered. Other grades show intermediate degrees of recovery.

Discussion

Since these were selected cases it cannot be said that the results represent the true average course of poliomyelitis paralysis. Indeed the amount of muscle weakness is so varied from case to case that it is almost impossible to arrive at "averages." What actually has been presented is the course of intermediate cases of paralysis; two total quadraplegic cases who were treated in respirators and made very little improvement and twenty-six very mild cases who recovered in the course of a few months have been left out. Furthermore, it is recognized that estimation of the power of individual muscles or muscle groups may not measure the actual improvement in general function; conpensatory action of the stronger muscles and adjustment of posture contribute to the improvement in ultimate performance.

We wish to emphasize that more than 95% of the treatment was given in a small rural hospital and in farm or village homes. No methods except standard physiotherapy (passive movements and re-education) were used. Early rehabilitation in the home environment and participation in treatment by other members of the family was insisted upon.

In the vast majority of cases the prognosis for recovery is good.

During the acute stage the patient should be treated as a case of acute encephalomyelitis; sedation and complete rest in the optimum position are imperative and any attempt to overcome muscle spasm by forced passive movement is strictly contraindicated. The patient must not be disturbed by hot fomentation, baths, lumbar puncture or splinting. Any contracture that may develop during this period is easily overcome when the acute inflammation in the cord has subsided.

When acute tenderness has disappeared and when the patient shows willingness to co-operate without distress in active re-education gentle passive movements within the limit of pain should be given once daily to those joints unable to perform full range of movement. Thereafter re-education of paralyzed muscles should proceed as in peripheral nerve injuries.

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Cancer

Tumor Clinic*

Nov. 17, 1953
Reported by Dr. D. W. Penner

Master T. D.—Presented by Dr. S. A. Boyd

This is an eight and a half year old boy—an only child. There is nothing significant about his previous history except as related to the present findings. On January 22, 1951, I saw him because he had complained of pain in the left hip. At that time he had some spasm and restricted movement of the hip, particularly on adduction. There was no history of any injury and we kept him at rest. On February 6, 1951, he still persisted in complaining of pain. There was no restriction of movement on walking. He was seen by Dr. A. Gibson whose note is as follows: "Slight resistance in all directions of hip movement—walks as if to guard the joint—in all probability traumatic". X-ray of the upper femur at the time was negative.

He passed over that incident and I think the following dates may be important. In May, 1951, I saw him with an acute otitis media, in June 1951, for an abrasion over the knee, on March 12, 1952, for an acute pharyngitis, and on April 30, 1952, with chicken-pox. There was no complaint on any of these occasions of any difficulty with walking or discomfort in the leg. I didn't see him again between that date and November 26, 1953. He had been at Detroit Lakes in the summer and was complaining of pain in the left thigh at that time. He states that it was rather constant, occurring every day, and that there was some swelling in the thigh which subsided. When I saw him in November, 1953—apparently it did not seem to disturb the parents too much—he had an obvious hard swelling in the left thigh. I think I can leave the rest of the history to Dr. C. Hollenberg who has been seeing him latterly.

Dr. Hollenberg—I saw this boy for the first time on November 30, 1953, with the history of pain in the lower part of the left thigh since about November, 1952. The swelling that he had has remained but the pain has been relieved. The week before I saw him he had been shovelling some snow during the day and that night was awakened with pain. He has been having recurrent pain ever since then. One important thing is that an aspirin would relieve the pain. As far as the laboratory findings are concerned, the blood count is normal, Wasserman negative, Sedimentation rate 40 mm., 46% lymphocytes. He had this swelling in the lower part of the femur extending not quite to the knee. On X-ray you could determine that this was a bony swelling and the soft tissues were not involved. The left leg is one inch longer than the right. On the basis of this I brought him to hospital for further investigation, mainly to get further X-rays and to do a biopsy. The biopsy was taken from an adjacent site, in consultation with Dr. Penner. The bone was very hard; it was just like chipping ivory. The medulla was very firm and sclerotic. There was no pus. The culture from it was subsequently reported as "no growth".

Dr. W. T. Dingle—(X-ray discussion). These are the first films taken in this hospital. You can see a thickening of nearly one-half of the shaft of the femur. It extends from about the midline of the bone almost down to the end of the shaft, but not involving the distal end. There is some subperiosteal formation of new bone. The cortex underlying this is quite thick. There is rather an irregular texture to this thickened cortex all through the involved area. There was some discussion in the Department about the probable diagnosis in this case and the two diagnoses considered most likely were Ewing's tumor or osteomyelitis which had been modified to some degree with antibiotics. The other diagnoses considered were osteoid osteoma, and this was thought possible but unlikely because there was no convincing evidence of a nidus present. It is also a larger lesion than one usually sees. Syphilitic osteolysis wasn't considered very seriously. Some thought it might be a complication of some sort from a leukemia or the like. I think the majority considered it a Ewing's tumor.

Dr. J. Lederman—This bone showed, as you might expect from the picture, considerable new bone formation, particularly in the cortex and some in the marrow cavity as well, associated with a fibrosis of the marrow and some round-cell or plasma-cell infiltration in this fibrotic tissue. I don't think any specific diagnosis can be made except that one can rule out primary bone tumors and malignant bone tumors in general. One interesting feature is, this marrow tissue which is so fibrosed is also exceedingly vascular, which might account for the extra growth and lengthening of that leg. One can conceive of this happening in a number of the conditions Dr. Dingle mentioned and in chronic osteitis, Garre's sclerosing osteitis, or osteoid osteoma. I should think that would be as close as we can get.

Dr. Bruser—The X-ray films and slides were discussed at Deer Lodge Hospital two weeks ago and the consensus of opinion was pretty much as Dr. Dingle mentions. The most considered diag-

*Presented at Thursday Luncheon by the Surgical Department of the Winnipeg General Hospital, with Dr. D. W. Penner acting as chairman.

noses were osteoid osteoma and Ewing's tumor. From where I am sitting here it seems there may be a nidus just below where the biopsy was taken.

Dr. Merkeley—Could this be an aneurysm of bone?

Dr. Penner—No.

Dr. Ferguson—I believe the condition called sclerosing osteitis of Garre is similar to this.

Dr. Hollenberg—The important thing is he was relieved by a single aspirin. My clinical diagnosis was osteoid osteoma or Ewing's, but when I first saw the X-ray films I could not see a nidus and decided on Ewing's tumor as the diagnosis. As Dr. Bruser says, it does seem as if there is a nidus present.

Dr. E. Black—Can you tell what you are going to do about this?

Dr. Hollenberg—The next thing, after having the sutures out, is to have further X-ray films and try to localize the nidus, and if one is found I would be inclined to remove it.

Case No. 2

Mrs. F. G.—Presented by Dr. F. W. Duval

This is a 56 year old woman with a long history of complaints involving her right upper jaw and nose. In 1936 she had a nasal polyp removed. In 1945 she complained of pain in the right side of her face and she was also complaining of excessive secretions from the right side of her nose. She wasn't seen again, as far as we can make out from that time until August 1953, when she was seen by a dentist for pain in the right maxilla which the dentist considered due to bad teeth. Three teeth were extracted from the right upper jaw then. This didn't appear to relieve the pain and she was seen then in the Outpatient Department of the Winnipeg General Hospital in November 1953. X-ray films of the right antrum and right upper alveolus were taken and the Doctor who saw her (Dr. Robert Black) considered she required treatment in hospital. On admission she gave a history of very severe pain, more severe than ever before, in the right upper jaw. There was marked oedema and swelling of the right face and some intraorbital edema. On admission she was admitted to the Ear, Nose and Throat service and has been looked after by Dr. Robert Black since then.

Dr. Dingle—(Discussion on X-ray films). There is not very much to demonstrate on these films. The one obvious finding is a very dense right antrum. If you could see more closely you could see some bone destruction. If this is a tumor it is involving the right side of the antrum and extending to the base of the skull involving the right pterygoid bone. The report on the initial films taken uptown is as follows: "The frontal sinuses are scarcely developed at all. There is some slight ethmoid cloudiness. The mucosal lining of the right antrum is markedly thickened. It does not contain any appreciable fluid. Laterally in the Zygoma there is a suggestion of bone destruction. The lateral margin of the right antrum in the vicinity is completely indistinct. No abnormality of the left antrum is shown. The sphenoid sinuses appear more dense than normal. Impression: A destructive lesion in the right zygoma, possibly inflammatory in nature, should be suspected. Dr. A. E. Childe." This film was taken November 25, 1953.

Dr. Robert Black—The only thing to see is the swelling on the zygomatic arch. It was quite tender on admission but is not so much so now. Most of the swelling you see now is from the surgical biopsy. The appearance of the films and the history led us to believe it to be a tumor. I opened it and was surprised to find that the antrum was practically normal. It looked like inflammatory hypertrophy and I took a biopsy including a piece of bone from the canine fossa and these were positive for epidermoid carcinoma. Usually such cases have a large fungating mass but she has not. A great deal of this cloudiness may be due to the surgical interference she has had.

Dr. Penner—The tissues all show infiltrating epidermoid carcinoma, grade 2. Dr. Merkeley, would you like to comment on treatment of this?

Dr. Merkeley—I think it is primarily a surgical problem and radiation can be used well in conjunction with the surgery. My treatment of choice would be a radical approach with sacrifice of the eye. She needs a radical maxilla done on that side and exenteration of the orbit. The principle in doing this is to leave a large, as large as possible, defect so it can be easily radiated if there are recurrences. I think you are better off to do this so you can see what you are radiating rather than have pre-operative radiation which may destroy the bone. The X-ray therapy doesn't have as good an effect pre-operatively as post-operatively. I don't think she is inoperable.

Dr. R. Walton—These lesions are frequently treated by a combination of surgery and irradiation. Where the lesions are smaller than this, the easiest way is to slip a radon tube into the middle, or as close to the middle as possible. You get in the neighborhood of the needle, some necrosis and then this can be removed by diathermy along with any other tumor-bearing fragments about four weeks after the radon implant. In a large lesion like this it is of no use and one is forced to use external radiation. Dr. Merkeley's remarks about producing radiation necrosis of bone used to be true but not now. You can, in fact, get away without any bone

necrosis with the new methods. The damage to skin is almost nil now with the cobalt beam. I would give a course of pre-operative radiation (cobalt), full dose, and then do a surgical removal later. I feel that is a better method than post-operative radiation.

Dr. Merkeley—What is done with recurrence after?

Dr. Walton—It is still possible to treat a local recurrence.

Dr. R. Black—About the possibility of removing this maxilla—Dr. W. Alexander and I have treated two or three similar ones in the past year. This growth seems, however, to have extended across the midline and probably back into the sphenoid and we think that surgical removal might not get all the tumor so I think x-radiation might be better in this case.

Dr. Merkeley—If you are going to do a radical maxilla you would remove the sphenoid, the ethmoid, and you leave the patient with a large orbital defect and skin graft. Then you can look in and see if there is any recurrence. Then you can radiate. She will have to lose the eye. I think that the results are pretty good, cosmetically. They can wear a black patch and the scar is practically invisible.

Dr. A. Klass—What effect on the eye would there be with radiation alone?

Dr. Walton—She would lose the sight of the eye.

Mr. R. I.—Presented by Dr. F. W. Duval

This man, at the age of 53, was admitted to this hospital in January 1935. The fact that drew his attention to the pathology in his mouth was that he had a lesion on the right side of his mouth which caused pain when wearing his dental plate. At that time it was noticed he had, on the opposite side of the lower alveolar margin, a painless lump which was described in the history as being about the size of a 5c piece. He had an operation for removal of both these tumors. The one on the right side was an abscess and the one on the left side—the non-tender lump—was an epidermoid carcinoma.

In January, 1936, he returned with a recurrence which was excised with diathermy and cauterized and the biopsy at that time confirmed epidermoid carcinoma. In March 1936 he returned with another recurrence which was again excised and cauterized. Dr. Penner has reviewed the pathology and will discuss this later. Dr. M. R. MacCharles treated the patient at the time and he will discuss the treatment further.

Dr. M. R. MacCharles—Dr. W. E. Campbell was in charge of the man at the time and I was just a very interested observer. These antral tumors are very interesting cases. If they grow from the base of the antrum they usually appear in the mouth. If they grow on the medial wall they often cause serous or purulent discharge from the nose. If they grow from the roof the first thing noticed is proptosis. If they grow from the anterior wall they present a swelling in the face. If they grow on the posterior wall they are often mistaken for a trigemenal neuralgia. One of the first cases I saw had been treated in Mayo Clinic for a tic douloureux and he had carcinoma of the antrum. The site of initial growth is what we usually base our treatment on. We have not been very successful in permanent cures. This is the oldest patient, cure-wise, alive and well, we have. Looking back at the treatment he had it certainly wasn't ideal according to our present standards. Over a period of two years he kept coming back with recurrences. I don't feel at all optimistic about treating these from the surgical standpoint. I don't think there is anything more to say about this. The one characteristic these tumors have is they are local problems and don't metastasize as a rule. They very seldom spread in the neck or blood stream, but the local problem is often insuperable. I think we have had others who have gone about five to ten years, but this man had gone nineteen years. I don't think this will ever recur again. I would agree with Dr. Merkeley that the defect in these cases is minimal and the cosmetic results fairly good and no hazard in treating them. I don't share his surgical enthusiasm for remote results. I think our cures are about 5% or less.

Dr. Merkeley—The surgical figures at Memorial Hospital for surgery with post-operative radiation are about 30% cures.

Dr. Penner—What can you achieve with radiation, Dr. Walton?

Dr. Walton—I think there is a group in Mayo Clinic using pre-operation and surgery and their figures are about 60%. Radiation alone is limited to small tumors and I think the figures would be about the same. The combination of the two, I think, is necessary.

Dr. Penner—Perhaps the reason we are seeing this man today is the low grade of the tumor. Dr. Warner questioned whether it was carcinoma; Dr. Lederman agreed it was a very low grade, adult type keratinizing squamous carcinoma.

Dr. Merkeley—I think you should take the whole bone out and then radiate.

Dr. C. W. Clark—What do you do about the skin flap?

Dr. Merkeley—You skin graft the whole area and radiate through the orbital or palatal defect.

Dr. R. Black—I thought the Americans had a very much higher cure rate than the English report.

Dr. Walton—I think the figures are about the same.

Miss N. T.—Presented by Dr. M. R. MacCharles

This lady, at age 53, was admitted to the Winnipeg General Hospital June 4, 1930, with an admission diagnoses of bowel obstruction. Following a barium enema which revealed a filling defect in the transverse colon she was operated on July 9, 1930, and the operative report is as follows: "The abdomen was opened by a right rectus incision about 5 inches long. The abdomen was explored and a large growth about the size of a fist was found in the transverse colon near the hepatic flexure. It also involved part of the ascending colon. The liver did not appear to have any metastases. Enlarged nodes were found in the transverse mesocolon. The stomach, gallbladder, pelvic viscera were normal. The preaortic nodes were not involved. The cecum, ascending colon, and about one-half of the transverse colon and the lower five inches of the ileum were removed and a side-to-side anastomosis performed between the lower end of the ileum and the remainder of the transverse colon. In removing the bowel enlarged nodes in the transverse mesocolon were removed with the mesentery."

We almost always have a few skeptics who believe you can't cure carcinoma. This lady has survived, tumor free, following carcinoma of the colon. This was not a small tumor, nor an early one, and it has a definite tissue diagnosis.

Dr. Lederman—The pathology report is dated July 10, 1930, and the total report is: Large ulcerated malignant growth completely encircling the cecum, producing marked narrowing but not complete stenosis. Microscopic: Adenocarcinoma grade 2."

This report obviously preceded the era of Duke's staging of carcinoma gastro-intestinal tract. Duke's staging has been of considerable prognostic value in assessing any specimens from the gastro-intestinal tract but it is particularly applicable to rectum and sigmoid, to a greater extent than cecum, at least. Stage 1 is when the tumor is not through the muscle or through the peritoneal coat and does not involve any lymph nodes; here there is about 90% chance of cure. Duke's B stage is when the tumor involves the serosa but doesn't extend into the lymph nodes; here there is about 60% chance of cure. Duke's C is when the regional extend into the lymph nodes; here there is about 10% chance of cure. We don't of course know the extent of this patient's tumor from the anatomical standpoint.

Dr. Penner—One thing that has always been a point of much discussion in our Tumor clinics is the value of occult blood in cases like this. The pathologists have always considered it to be very useful and it is a very inexpensive test. I would like to ask Dr. Pearlman what he thinks of the value of occult blood in stool as a screening procedure for gastro-intestinal neoplasms.

Dr. I. Pearlman—It all depends, naturally, what test is used. Some tests are too sensitive. The Guiaic test is a fairly good one. The occult blood if positive, with some anaemia, should point to some further investigation. I think, as a screening test it should be done almost all the time, especially in people over 50 years of age. It is cheap and doesn't take much time and if positive, is of some value. I had a woman about a month ago with no gastro-intestinal complaints. Her hemoglobin was below 50% and definitely, occult blood test was positive. She had no vomiting and no loss of weight. A little edema in the legs was the only thing she showed. Barium series following the positive occult blood showed a tumor in the stomach. It proved to be adenocarcinoma which had penetrated through the mucosa. The Lahey people think it is a good screening test, not a diagnostic measure though.

Dr. F. A. Mathewson—I think that unless the test is strongly positive it is rather disappointing. Frequently you find it in small quantities. I feel it should be checked and re-checked though, and people should be put on special diets and have it re-checked again to be of real value.

Dr. Klass—I think the best comment I have heard on this is it gives a 60% degree of accuracy and is 10% better than tossing a coin.

Dr. MacCharles—I think the occult blood is a good test. I think that in all these tests, positive reports are of some value and negative ones don't mean anything. In the absence of any other positive findings I don't worry too much about it.

Fugitive Pieces

J. C. Hossack, M.D., C.M. (Man.)

De Herpetibus

Snakes, taking them by and large, do not greatly interest me, but there are times when I am compelled to give them some attention.

These times are, specifically, when I come across some insigne in which a snake appears. Our profession has a considerable number of such insignia since every Society or Association possesses one and each must be different from all the others.

My present concern is with those serpents which, as it were, work for us. Of these there are several, the senior member of the group being the one employed by the C.M.A. He is a bulgy, droopy sort of beast which, in defiance of all the laws of physics, succeeds in clinging to a wand-like rod which he barely touches. If there is any symbolism here it is that doctors grow pot-bellied and hump-backed in their efforts to accomplish the impossible.

Younger by nearly sixty-five years is the M.M.A. representative. Despite his comparative youth he is if anything more kyphotic and lordotic than his pappy. Moreover his head is smaller and his belly is bigger whatever that may mean. Like his sire he also manages to maintain an impossible position. No snake yet created, not even "Th' infernal serpent, he . . . whose guile, deceiv'd the mother of mankind," could manage to do what we see in our Association ophidians.

Now, if you cast your eye upon them, you will find that both these creatures bear a nasty resemblance to the dollar sign. In fact there are two dollar signs, both of them reversed so as not to be too obvious. There may be a meaning in this because two bucks was considered a decent fee when these reptiles first took up their duties.

Next in order of time comes the Winnipeg Medical Society Specimen which, alone of all his fellows, clings naturally, comfortably and physiologically to a sensible staff such as Aesculapius would not have scorned to use. You are indebted to Tony Gowron for the reptile and to Cicero for the motto associated with it—absit invidia, "Let ill will be absent."

The M.M.A. snake is also fortified with a motto and it is a most excellent one. It is condensed into two words in Latin—"Quaecunque vera," "Whatsoever (things are) true." Extended to its full length and put into English we find that it is St. Paul's advice to the Philippians—Whatsoever things are true, whatsoever things are honest, whatsoever things are just, whatsoever things are pure, whatsoever things are lovely, whatsoever things are of good repute; if there be any virtue and if there be any praise, think of these things." Such at least it seems to me is the meaning the originator of the abbreviation meant it to have. In the days when that motto was chosen doctors knew more about both Latin and the Bible than they do now, and then a couple of Latin words were enough to bring the well-remembered passage to the memory and to the lips and, let us hope, into practice as well.

The most recent addition to the Circle of Medical Serpents is the one which serves the Manitoba Medical Service. The like of it is not to be found anywhere in the heavens or in the earth or in the waters that are under the earth. If one wonders how Ophidius C.M.A. or Oph.M.M.A. manage to keep their places, that wonder is redoubled when he views Oph.M.M.S. The whole device looks as if the inspiration for it had been a cork screw.

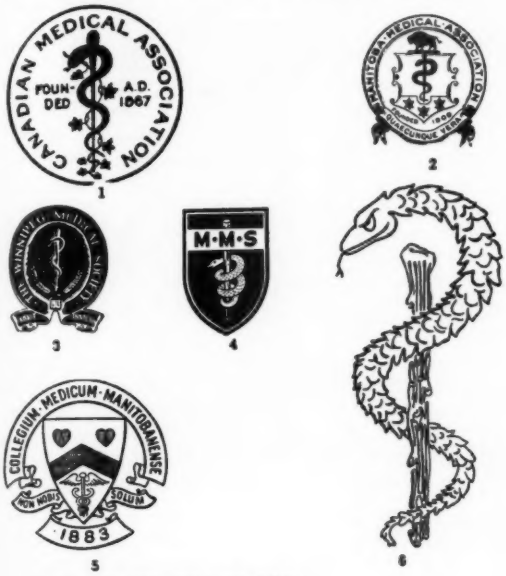
Ophidius M.M.S. utters no salutary admonition. It simply rears a sign bearing its initials. How these letters may be interpreted will depend upon the interpreter. A satisfied customer is liable to say they mean "Most Magnificent Service." A Scot might read into them "Much Money Saved"; while a dissatisfied medical member will suggest that the Trustees read them as "More Money, Sirs." Meanwhile Ophidius Ememessicus, completely defiant of all the laws of Nature, "makes like a cork screw" suggesting, no doubt, that if people were to take a little wine for their stomachs' sake and their often infirmities (or perhaps, leave the stuff alone), they would get along with a good many fewer visits to the medical members.

With all the foregoing varmints you are familiar. Let me now introduce to you one that will be to many a complete stranger. This is Ophidius Ferox et Horrendus—the fierce and frightful snake. He is the creation of a young colleague (Dr. Thorson) who was an undergraduate at the time of this creature's conception. He, (O.F. et H), is employed by the University of Manitoba Medical Students' Association and his job is to decorate their journal. I would like you to take a glance at him then soothly swear, was never beast so fierce and "quare."

Here is no smoothie! Here is no droopy, dejected, pin-headed, pot-bellied, kyphotic, lordotic defier of the laws of Nature! He is a rough, tough reptile with the gleam of battle in his eye—an eye, incidentally, like Mars' that threatens and commands. His scaly coat is not to be grasped by the bare hand or one sheathed in a silk glove. He is not to be petted or soothed by gentle strokings.

Moreover, apart from being firmly in contact with a very substantial staff he rears his head above it and glowers about him as if for signs of approaching danger. Here is a snake as is a

snake! Here is a monster worthy to serve as our device! Indeed, I would go so far as to say that we should adopt him as our exclusive representative so typical is he of the Medicine of Today.



1. Canadian Medical Association.
2. Manitoba Medical Association.
3. Winnipeg Medical Society.
4. Manitoba Medical Service.
5. Manitoba Medical College.
6. University of Manitoba Medical Students' Journal.

In the old days the members of our profession were held in awe by the hoi polloi. They were so potent of harm as well as of good that their reputation afforded them protection. Attempts at their government were usually unsuccessful for they practised a mystery which they alone comprehended, and the conduct of which no others were competent to control. Their governance, therefore, lay with themselves.

But such is not the case today. Our independence is being threatened; our authority is being assailed; our mystery is being explained to anyone who has a penny wherewith to buy a magazine of any sort. Our smooth and sleepy serpents can no longer protect us. We must gird our loins for battle and wage war against those who threaten to denude us of our secrets and to deprive us of our privileges. And under what banner are we more likely to be successful than that which bears the strange device of Ophidius Ferox et Horrendus? As the thistle is among plants so is O F. et H. among animals, and natural to both is the same motto "Nemo me impune lacessit"—"No one can monkey with me and get away with it!"

So much for single snakes. On the coat of arms of the Medical College we find two snakes entwined about a winged rod. This occupies the base of the shield, the field of which is divided by a "bend." Above the bend are two hearts, one whitish and the other definitely blackish. As our craft is divided into two main branches it is possible that one heart is given to represent each. In this case the logical assumption is that the white heart stands for physic and, there being left only one other department and one other heart, the blackish heart must represent surgery. I don't say that such was the intention of the artist but there must be some explanation and I can find no other more reasonable. (I wonder if my arch-critic Dr. Adamson will agree to that!)

Now, there are not only two hearts but also two snakes. Can it be that the snakes also represent physic and surgery? If so then why the fond embrace? Does it represent the amity that should exist between those who loath and those who love the shedding of blood? Or is there a deeper, darker meaning? Could it be—oh horrors!— of course not!—but could it be that the two snakes, one a physician and one a surgeon, have just completed—perish the thought, doctors don't do such things—but mightn't it just be within the bounds of possibility that they had just completed that infamous operation called (sob) dichotomy? and are hugging themselves for doing it? Pro pudor! Oh Shame, where is thy blush!! And on the College bearings at that! What a terrible example for innocent and callow youths!

What makes this awful interpretation more likely is the fact that two snakes entwined about a winged rod is the caduceus—an accoutrement of Mercury, not of Aesculapius. And Mercury, sly, greedy, plausible, dishonest, money-loving, mendacious, unscrupulous Mercury, was the god of thieves, merchants, defrauders and the like.

His only contact with medicine (apart from his use as a mineral) is the fact that he obtained his rod from Apollo. What happened after the acquisition of the rod and how Mercury came to be used in the treatment of lues are interesting stories for which we have no space here.

Any time that the caduceus has been accepted as a medical emblem it has been either out of ignorance of its true significance or with tongue in cheek. The United States Congress chose it for their Army Medical Corps because it was "more artistic" than the Aesculapian Staff and Snake! The Senators were warned of its meaning but ignored the warning.

It was for a while adopted in England during the reign of Henry VIII. The King's physician, William Butts, and his royal master were both excellent classical scholars (Henry was a translator of no small merit) and were well aware of what they were doing. The Court physicians were then on notoriously friendly terms with the Mammon

of Unrighteousness. In other words they were, many of them were out-and-out racketeers. The substitution of the caduceus for the symbol of Aesculapius was a broad hint as to the god they served.

Why Snakes?

While on the subject we might consider how the snake came to be adopted as a medical emblem. The exact reason is nowhere given. Taking it by and large, however, primitive peoples in every age and clime have held sacred two classes of creatures—the especially attractive and the particularly repellent. In every part of the world where they are known snakes have been objects of worship. Sometimes they were venerated as the personification of wisdom ("wise as the serpent") sometimes held in awe as the embodiment of evil ("th' infernal serpent"). The fact that the creature each year sheds its skin led to the belief that it never died and so it became a symbol of immortality. Again by putting its tail in its mouth, it forms a circle, typical of endlessness and therefore of immortality.

There is a legend that Aesculapius was a serpent which became transformed into a human being, and another that the Temple of Aesculapius attracted snakes which served the god as assistants and cured the sick by licking them. When the Romans sent envoys to Epidaurus with an invitation to Aesculapius that he should visit their city and stay the plague, a great serpent was seen to convey itself on board and this was thought to be the god.

Zoologists have set aside a species of snake to which they have given the name Coluber Aesculapii or Elaphis Aesculapii.

The staff is properly a stout and knotted one, upon it the lame can lean and by it the weak are supported. Its roughness symbolizes the hard nature of the problems which the physician must solve. The correct emblem shows a snake physiologically applied to its support. The thin rods in our devices belong to Mercury.

The Snake in Treatment

During their wanderings in the wilderness the Children of Israel were attacked by fiery serpents so called by reason of the burning fever caused by their bites. "And much people of Israel died." To stay the deaths Moses was commanded to make a "fiery serpent, and set it upon a pole and it shall come to pass that every one that is bitten, when he looketh upon it, shall live."

The reasoning behind this therapeutic procedure is profound and complicated. Reduced to its simplest elements and without explaining the intervening reasoning it may be said that the revelation of a demon to itself is sufficient to cause its expulsion. The bite was poisonous because the demon entered with its venom and the evil spirit could be driven out only when it saw that it had been discovered. The demon, in other words was potent only so long as its identity was concealed.

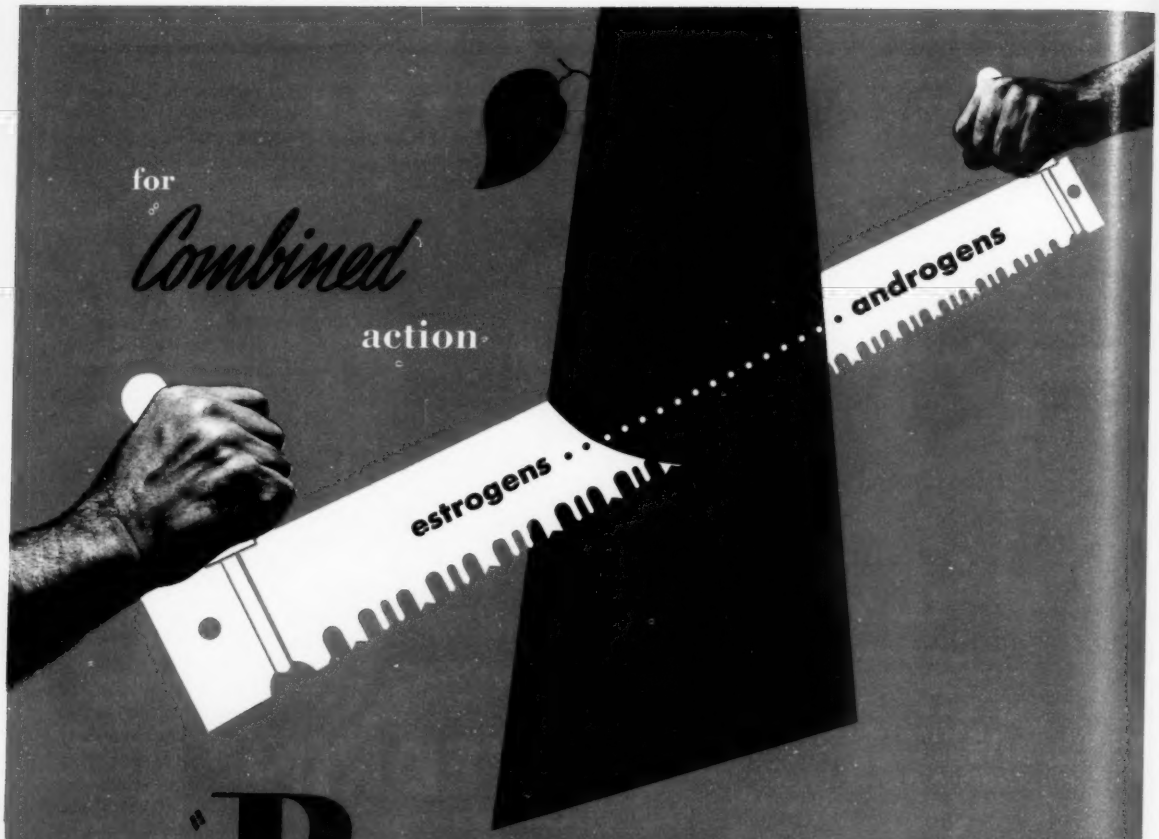
It is a very wide spread belief that what can in one form injure can, in another fashion, cure. This is essentially the principle of vaccination. In the case of snake poisoning the application of the dead snake to the wound it had inflicted was held to be curative. So was the inunction of viper-fat although the specific nature of this substance is somewhat put in question by Sir Richard Meade's statement that common oil can be used if the snake cannot be caught, killed and rendered.

Vipers particularly but other snakes as well were an essential constituent of Theriac—that strange hodge-podge of remedies which was regarded as **the** panacea. Its name still lives in "treacle" which was one of its ingredients. Theriac persisted in the British Pharmacopoeia, until Heberden's time when he, by a solitary majority of one, persuaded his colleagues to exclude it.

Theoretically theriac was not an irrational remedy. The ancients from Galen until the mid seventeenth century brought under the heading of "malignant diseases" all those toxic conditions due to vegetable, animal and mineral poisons which were accompanied by fever. As many of the symptoms were the same in most cases it seemed logical that a universal antidote could be found; and the supreme toxicity of poisonous snakes indicated their flesh as an important ingredient.

In Salmon's Dispensatory ((1696) we find this: "Essence of vipers is a most excellent medicine, dissolves all excrements and coagulation of humours, cleaning and purifying like soap; carrying out every ill by urine, sweat, or insensible transpiration, curing all sorts of gouts, the stone in both reins and bladder, leprosy, French Pox, scurvy, melancholy, all obstructions and putrefactions, loss of strength, decays of nature and consumption; so that, as it were, it even renovates the man, by taking away what is contrary to nature and adding what is requisite."

Renovating the man . . . taking away what is contrary to nature . . . adding what is requisite . . . Isn't that our job? And is not, then, the snake our proper creature? But he is the Agathodaemon, the giver of wisdom and healing, of happiness and good fortune, set firmly upon a stout staff, not a droopy monstrosity looking like a couple of bucks on a pencil. Such distortions are derogatory to his dignity and to ours. When next we devise an insigne let the snake upon it be worthy of himself and us.



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Medical History

The Golden Age of Anatomy in Edinburgh*

I. Maclaren Thompson
B.Sc., M.B., Ch.B. (Edin.), F.R.S.C., F.R.S.E.†
Winnipeg

Before the Dawn

Anatomical dissections were authorized in Edinburgh as early as 1505, long before there was any organized School of Medicine there. Naturally, the surgeons were the pioneers in the practical study of Anatomy. A century or so later they and the Town Council were the pioneers in organized medical education in Edinburgh, the early 'Professors' of Anatomy being 'extra-mural' part-time surgeon-anatomists.

The first Professor of Anatomy in the University was the first of the famous Monro dynasty, father, son and grandson, Alexander Monro **primus, secondus** and **tertius**, who between them occupied the chair of Anatomy for the astounding period of 120 years, from 1726 to 1846. All three Monros were part-time anatomists; indeed, they professed to teach both Anatomy and Surgery. Monro I (1697-1767) earned a fine reputation as an anatomist, and is remembered for his part in establishing the Faculty of Medicine of the University, the Royal Infirmary, and one of the precursors of the Royal Society of Edinburgh. He heralded the dawn. Though Monro III (1773-1859) actually lived in the Golden Age, he was not of it; according to Sir Arthur Keith, 'He failed in the first duty of a professor, the duty of bringing students in touch with the best movements of the time'.

The Golden Age

I regard the Golden Age of Anatomy in Edinburgh as beginning with the appointment of Alexander Monro II as Professor of Anatomy in the University in 1758 (he had been joint professor with his father for the previous four years), and ending with the death of Goodsir in 1867, a period of about a century. The brightest planets in the Edinburgh anatomical firmament of those days were Alexander Monro II, Robert Knox and John Goodsir. These, together with the lesser luminaries clustered round them, constituted an anatomical galaxy still worth contemplating. Monro II and Goodsir were professors in the University, their incumbencies being separated by that of Monro III; Knox was lecturer at Surgeons' Hall.

Alexander Monro II (1733-1817)

Monro I trained his son to be his successor; in 1754 **Secundus** was appointed joint professor with his father. Leave of absence was spent in Europe, partly studying Anatomy under the elder Meckel in Berlin. He is also said to have attended the anatomical lectures of William Hunter in London. The resignation of his father in 1758 left **Secundus** sole occupant of the chair, which he adorned for the next half century, retiring in 1808 in favour of his son, Alexander Monro III, who had been joint professor for the previous ten years.

The reputation of Monro II as a teacher of Anatomy was even higher than that of his father, and contributed much to the growth of the Faculty of Medicine, 'his elegant and scientific lectures attracting students from all quarters of the globe,' according to an obituary notice.

But it was his investigations that began the Golden Age of Anatomy in Edinburgh. His thesis was entitled, in the universal language of scholarship, **De Testibus et Semine in Variis Animalibus;** it was said to be 'fully twice the size of ordinary theses'. Under Meckel he learned how to inject lymphatics and other minute tubes with quicksilver, and laid the observational foundations of an essay, **De Venis Lymphaticis Valvulosis** (1757), in which he described the lymphatic vessels as opening out of the connective tissue spaces, and considered them to be 'absorbents.' William Hunter discussed the same two points in his lectures, and Monro accused Hunter of having pilfered these ideas from him. One of the famous medical controversies of the eighteenth century ensued. I shall simply quote the conclusion of a modern medical historian, J. D. Comrie, who has studied the available evidence; referring to Monro, he writes: 'On the balance of probability, the original discovery that the lymphatics form an independent absorbent system is really his.' One of Monro's distinguished pupils was William Hewson (1739-74), who subsequently worked with John and William Hunter in London; he died in consequence of a dissection wound. In 1771 Hewson published an excellent experimental investigation on the clotting of blood; and in the year of his death appeared his famous **Description of the Lymphatic System in the Human Subject and in Other Animals.** Again Monro claimed priority, this time in describing lymphatics in lower vertebrates; and again here is Comrie's judicial summing up: 'It is quite clear that Monro had shown injections of the lymphatics in these animals to his class before Hewson became a medical student, but he certainly never described and figured them with the fullness and accuracy of the latter's work.'

In 1783 Monro published his **Observations on the Structure and Function of the Nervous System.** In this he wrote:

> 'So far back as the year 1753, soon after I began the study of Anatomy, I discovered

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†Professor of Anatomy and Chairman of the Department, University of Manitoba.

> that the lateral ventricles of the Human Brain communicated with each other, and at the same place, with the Middle or Third Ventricle of the Brain . . .'

This is, of course, the historical basis for the familiar term, foramen of Monro, now designated more descriptively the interventricular foramen. Monro's attention was drawn to the foramen in the brain from a patient of Dr. Robert Whytt (1714-66), one of the most brilliant of eighteenth century physicians, a pupil of Monro I, and Professor of Medicine in the University of Edinburgh. Though not a professional anatomist, he made outstanding contributions to our understanding of the anatomical basis of reflex actions, showing, for instance, that only limited parts of the central nervous system are essential for certain reflexes, e.g. those of the pupil. The patient under discussion suffered from what Whytt called 'Dropsy in the Brain,' and it was the hydrocephalically enlarged foramen that Monro noticed. This was one of the cases upon which Whytt based his original account of tuberculous meningitis. I have not looked into the matter, but it does seem unlikely that not one of Monro's predecessors, Galen, Vesalius, and all the rest, ever noticed or mentioned this obvious communication between the ventricles. Of course the observation was original with Monro, and certainly he seems to have been the first to appreciate the significance of the foramen.

In 1785 Monro published his **Structure and Physiology of Fishes explained and compared with those of Man and other Animals,** which established his European reputation as a comparative anatomist. The appearance three years later of his **Bursae Mucosae of the Human Body** marked him as a distinguished human anatomist, for his was the first systematic account of these surgically important little structures. His last book, **Three Treatises on the Brain, the Eye and the Ear,** was published in 1797; and he retired in 1808. He enjoyed an extensive practice as a physician.

John and Charles Bell

These famous brothers, John older than Charles, make us think of William and John Hunter. All four were Scotsmen; all figure in the history of Anatomy, as well as in that of other branches of Medicine. Both the Bells were pupils of Monro II.

John Bell (1763-1820) was one of the leading Edinburgh surgeons of his day. Although the Monros were clinical practitioners, they presented Anatomy somewhat formally and pedantically, after the European fashion of Winslow and others. For initiating the teaching of Anatomy in its most intimate relaionships with the rest of Medicine (represented in this instance by Surgery), Edinburgh has to thank John Bell; he was the founder of the Edinburgh School of Surgical Anatomy. He conducted an extra-mural class in Anatomy for about a decade; latterly he was assisted by his brother Charles. In 1794 John Bell published his **Engravings of the Bones, Muscles and Joints;** these exemplify two things: the 'applied' point of view in teaching Anatomy, and artistic talents suggesting the more widely known artistry of his brother. In Garrison's opinion, this 'is one of the milestones in the history of anatomic delineation.' Bell's **Anatomy of the Human Body** appeared in three volumes between 1793 and 1802. The section on the nervous system was contributed by Charles, who edited the later editions. I consider this a truly great work in the field of Dynamic Human Anatomy. John Bell was an authority on the vascular system. In the preface to the sixth edition (published after John's death) Charles wrote:

> 'Of the first part of the work by my brother, I may speak more freely . . . It will not soon be surpassed in correctness and minuteness of description.
>
> I have not dared to touch the History of the Arteries, as delivered by my brother; the rapid improvement in the surgery of the arteries, which followed as a consequence of the first publication of this part of the Anatomy, has, with me, made it sacred.'

The 'improvement in the surgery of the arteries' referred to was based largely upon Bell's extensive work on anastomoses, and his application of this knowledge to the saving of life and limb by ligating instead of amputating. This vital matter was extended in Bell's **Discourses on the Nature and Cure of Wounds** (1795), and in his **Principles of Surgery** (1801-7). John Bell's great merits have been obscured by the glamour surrounding his brother, Sir Charles, and his (John's) counterpart a generation later, James Syme; it seems to me that John Bell has received less than his share of credit both in his own day and since.

Sir Charles Bell (1774-1842) concerns us here much less than his older brother, for almost all his outstanding work was done in London. A student of Monro II, and assistant to his brother in teaching Anatomy, he took charge of the class (extra-mural) for five years, moving to London in 1804. Thirty-two years later he returned to Edinburgh as Professor of Surgery, but his great days were over. Before leaving Edinburgh Charles Bell published several illustrated works on Anatomy, including a beautiful set of **Engravings of the Brain and Nervous System** (1802). Two years after he went to London his **Anatomy of Expression** appeared, a splendid investigation in Dynamic Anatomy, illustrated with beautiful engravings; most of this work had been done in Edinburgh. Of the rest of Bell's famous work, all that can be said here is that it reflects in part his early training during the Golden Age of Anatomy in

Edinburgh. About 1825 Bell's museum was purchased by the Royal College of Surgeons of Edinburgh, where it remains to this day; it includes some interesting water-colors (by Bell) of wounds received at the battle of Waterloo.

John Barclay (1758-1826)

John Barclay was a licensed preacher who turned to Medicine, studying Anatomy under Monro II, and, for a brief period after graduation, in London. Returning to Edinburgh, he was for a short time anatomical assistant to John Bell; then he embarked upon an anatomical course of his own, which coexisted first with that of John Bell, then with that of Charles. The departure of the latter from Edinburgh left Barclay the sole extra-mural anatomist, his course being officially recognized by the College of Surgeons, and being given in a building close to Surgeons' Hall. Barclay never engaged in medical practice; he was the first full-time anatomist. His interest had evidently been directed by Monro II toward Comparative Anatomy, for this he cultivated enthusiastically: he published upon it, he gave a special course in it, and he used it to enhance the effectiveness of his expositions of Human Anatomy. At one time a movement was started to create a chair of Comparative Anatomy in the University for him, but nothing came of it. That Barclay did not neglect the Dynamic Anatomy of the Bells is seen in his book **The Muscular Motions of the Human Body** (1808).

After the retirement of Monro II in 1808, the uninspiring teaching of Monro III drove most students to Barclay's lectures, where they benefitted from a gifted and enthusiastic full-time teacher. Sir Robert Christison, who was one of his students, has left us this pen-picture.

> 'His whole life and soul were in his profession, and with his students. He delighted to spend the whole day in his museum, dissecting-rooms, or lecture-hall, working with his pupils and assistants as diligently as the best of them. As a lecturer, he was all fire and zeal, and intent that his students should learn.'

Among Barclay's distinguished pupils may be mentioned: Robert Liston, the famous surgeon; William Sharpey, of whom more presently; Sir Richard Owen, one of the greatest of British comparative anatomists; and Robert Knox, who became Barclay's successor.

Robert Knox (1793-1862)

One of the most remarkable of anatomists, Robert Knox was born in Edinburgh in the year of John Hunter's death. Like so many other students of the time, he forsook Monro III for Barclay, who inspired him with a fiery zeal for Anatomy. He graduated M.D. in 1814, his thesis being **De Viribus Stimulantium et Narcoticorum in corpore sano.** The next year he was commissioned assistant-surgeon in the army; like Sir Charles Bell, he attended the wounded in Brussels after Waterloo; subsequently he served for several years with considerable distinction at the Cape of Good Hope. In 1821 Knox obtained a year's leave of absence from the army, ostensibly to improve his medical knowledge by post-graduate study in Europe. Actually, he spent most of his time studying Anatomy in Paris, especially under the great Cuvier and St. Hilaire. It seems probable that, after a latent period, Knox was now reacting to the anatomical stimulation of Barclay. At any rate, he evidently decided to become an anatomist in Edinburgh, but an anatomist of the French school. So to Edinburgh he returned; having no appointment, he settled down to several years of private research and writing, mostly on Comparative Anatomy, reading his papers before sundry scientific and medical societies in Edinburgh. In 1824 Knox was appointed in effect (though not in words) Conservator of the Museum of the Royal College of Surgeons of Edinburgh. He immediately put the museum 'on its feet'; this was crowned, in the following year, by the success of his efforts to secure the purchase by the College of the private museum of Sir Charles Bell, who was then about to transfer from the Windmill Street School of Anatomy to the Middlesex Hospital.

This museum work brought Knox into contact with his former teacher, Barclay, then nearing threescore and ten, and concerned about a successor. So in 1825 Knox became a teaching partner with Barclay, and in 1826 he was officially appointed Conservator of the entire College of Surgeons Museum. In August of that year Barclay died, leaving his private museum to the College of Surgeons, and leaving Knox the sole extra-mural anatomist.

It was also in 1826 that, for the first time in Edinburgh, dissecting by students was made compulsory. I do not know what part Knox may have played in bringing this about, but he was most unexpectedly and seriously involved in its consequences. For there was then no adequate legal supply of bodies to dissect, and grave-robbing (no new thing) increased, culminating in the Burke and Hare murders in 1828. Some of the victims, if not all, landed in Knox's establishment, and he was strongly suspected of guilty knowledge, to say the least. But no evidence against him was forthcoming, so no charge was laid. However, his personal reputation suffered irreparable damage, that not even he could live down. His less desirable personal characteristics (bombast, arrogance, quarrelsomeness) harmed him and helped his many enemies. His popularity and his classes declined until in 1845 he left Edinburgh. After some peripatetic lecturing and writing, Knox finally settled down (if the term be applicable to

such a man) to medical practice in London, where he died in 1862.

Knox was not a man easily delineated in a thumb-nail sketch such as this. His literary and research output was quite beyond even summarizing here. He laboured in all the fields of Anatomy popular amongst the anatomists of his day—or rather of his best days—except, perhaps, Experimental Anatomy. He was most outstanding in Comparative Anatomy and in Ethnology. Though no great discovery was his, he contributed more to anatomical knowledge than is commonly remembered today. As two instances, I may mention his demonstration of the muscular nature of the ciliary muscle of the eye (the muscle of accommodation), and his discovery of the macula lutea, previously considered a mammalian feature, in the eyes of certain lizards.

But it was as a teacher that Knox reigned supreme. Judging from the accounts that have come down to us from some who heard them, his best lectures must have been fascinating and stimulating to a degree somewhat difficult for us to understand; certainly they attracted large audiences, by no means restricted to the members of his class, nor even to the medical profession; and they aroused tremendous enthusiasm. So far as I can judge, his only rival in this regard was Josef Hyrtl (1810-94), who was appointed Professor of Anatomy in the University of Vienna the year before Knox left Edinburgh. Though not a truly great anatomist, Knox was certainly a brilliant one.

The best evidence of Knox's powers of stimulation consists of the long list of his pupils and assistants who distinguished themselves. These included Sir William Fergusson, the famous surgeon, and several anatomists, notably John Reid and John Goodsir.

John Reid (1809-49)

Reid was dissecting in Knox's establishment at the time of Burke and Hare. He graduated in 1830; and he showed his high opinion of Knox by becoming his demonstrator for three years (1833-36). Then he became extra-mural lecturer in Physiology, and pathologist to the Royal Infirmary. In 1841 he was elected Chandos Professor of Anatomy and Medicine in the University of St. Andrews. His death a few years later, after suffering dreadfully from cancer of the tongue, cut short a career of great promise.

All Reid's important researches were done in Edinburgh. The chief of them lay in the following fields: the placental circulation, the dynamic anatomy of the heart, and the nervous system. It was in the last that he was at his best. Inspired by the work of Sir Charles Bell on the spinal nerves, Reid, by a series of brilliant experimental studies, elucidated the dynamic anatomy of those most difficult of cranial nerves, the glossopharyngeal-vagus-accessory complex. He was an experimental anatomist of the lineage of Galen, Harvey and John Hunter. Though Reid's illness and early death limited the total volume of his researches, their quality entitles him to a place of honor in the Golden Age of Anatomy in Edinburgh.

Microscopic Anatomy

Following the epoch-making investigations of the fathers of Microscopic Anatomy, notably Malpighi (1628-94), the technical limitations of the early microscopes retarded the rate of progress in this field throughout the eighteenth century, nor did any great theoretical principle vivify the subject and instigate research. But the early years of the nineteenth century saw three movements that revived the use of the microscope in Anatomy. The first was the development of what was called General Anatomy, especially by the French anatomist Bichât and his pupil Béclard; this dealt with the minute anatomy and properties of the tissues and structures found widespread throughout the body—bones, fasciae, membranes, vessels, nerves, and the like. A second stimulus to anatomical microscopy came from the embryological discoveries of such European anatomists as J. F. Meckel, von Baer, and Rathke. The most powerful stimulus of all, however, came in 1838-9 with the popularization of the cell doctrine by Schleiden and Schwann. Two years later, in his **Allgemeine Anatomie**, Jakob Henle (1809-85) founded modern Histology upon a cellular basis. Nor must we overlook an important technical advance, the great improvement in achromatic lenses published in 1830 by Joseph Jackson Lister, the father of Lord Lister.

The older French brand of General Anatomy was taught in Edinburgh to some extent by the Monros, Barclay, and one or two others; but Knox, who was an anatomical Francophile, carried it much further. For the use of his students he translated **Béclard's Eléments d'Anatomie Générale** in 1830, adding an original Introduction and two Appendices. The resurgence of Microscopic Anatomy in the early nineteenth century was advanced in Edinburgh by a brilliant group, to which we must now turn.

One of Barclay's students who helped to make anatomical and physiological history was **William Sharpey** (1802-80). He obtained the diploma of the College of Surgeons at the age of 19; thereafter he spent some months studying Anatomy in the private anatomical establishment of Joshua Brooks, in London. Then followed several **Wanderjahre** in Europe, with intervals in Edinburgh. From 1831 to 1836 Sharpey conducted an extra-mural course in Anatomy in Edinburgh, and pursued his investigations, chiefly on ciliated epithelium and ciliary mechanics. His other chief researches were on bone, the term Sharpey's fibres being still in use. In 1836 Sharpey was appointed

Professor of Anatomy and Physiology in the institution then called the University of London, but now known as University College, where later on one of his students was Joseph Lister. This was a newly created chair, in which, for the first time, Microscopic Anatomy was associated with Physiology, leaving the Professorship of Anatomy, to which Richard Quain was appointed, to deal only with Gross or Macroscopic Anatomy. This transfer of Microscopic Anatomy from the rest of the subject to Physiology had far-reaching effects upon the history of British Anatomy and Physiology, and has been undone only in recent times; but that is not our present subject.

Thomas Wharton Jones (1808-91) is one of the oddest characters in British Anatomy. But my mention of him must be brief, for the same reason as in the case of Sir Charles Bell. Having studied Medicine in Edinburgh, Wharton Jones was one of Knox's assistants at the time of the Burke and Hare scandal; consequently he shared in the opprobrium associated therewith, and soon he left Edinburgh. After an unsettled decade, during which he did much research in various places, Wharton Jones was appointed lecturer in Anatomy, Physiology and Pathology at the Charing Cross Medical School. There one of his students was Thomas Henry Huxley, whose very first piece of research (on the sheath of the root of a hair) was encouraged and supervised by Wharton Jones. In 1851 he was appointed the first Professor of Ophthalmological Medicine and Surgery at University College, where one of his students was Joseph Lister; Wharton Jones also helped him with his first investigation, into the sphincter and dilator muscles of the pupil. To few men is it given to start on their investigational careers two such pupils as Huxley and Lister. The brilliant investigations that made Wharton Jones famous were all made away from Edinburgh, so they need not even be summarized here. They were in the fields of embryology, haematology (he discovered the amoeboid movements of leucocytes), the circulation, and inflammation. Lister's pre-antiseptic researches on inflammation show the acknowledged influence of Wharton Jones's work. Like Sir Charles Bell, Wharton Jones was an important 'product' of the Golden Age of Anatomy in Edinburgh. He waged bitter feuds with many people, notably Sharpey and Martin Barry, concerning both of whom he quite ignored (even in print) the admonition **De mortuis nil nisi bonum!**

Martin Barry (1802-55) is not easy to fit into this account of the Golden Age of Anatomy in Edinburgh, yet he unquestionably has a place here. He must have been endowed with private means, for he studied Medicine not only in Edinburgh, but also in London, Paris, Berlin, Erlangen and Heidelberg! At the latter University Tiedemann stimulated Barry to take an interest in Embryology, which he did to the end of his days. Graduating M.D. (Edin.) in 1833, he spent the next nine years between assiduous microscopic research, mostly (if not entirely) in Edinburgh, and tours of European centres, where he came under the influence of many of the leading microscopists and embryologists. Leaving Edinburgh in 1842, he lectured on Physiology at St. Thomas's Hospital, London, for a short time; then came a period as house surgeon at the Royal Maternity Hospital in Edinburgh. Several peripatetic years followed, divided between Europe and England, but without any regular appointment; in 1853 he settled in Suffolk, and he died two years later.

His life was devoted to microscopical research, chiefly in Embryology. The period of his Edinburgh researches comprised, roughly, the decade 1833-43, during which, as we have already seen, three major factors in the development of Microscopic Anatomy came into operation: (1) the availability of improved achromatic microscopes, (2) the establishment of the cell doctrine, and (3) the consequent founding of Cellular Histology and Embryology. Utilising the first, Barry was in the thick of the last two. He was the first microscopic embryologist of note in Edinburgh, if not in Britain. His chief Edinburgh researches were published in four memoirs by the Royal Society of London between 1839 and 1843, and earned for him the Royal Medal of that Society; they may be summarized thus. (1) He was the first to see spermatozoa inside the zona pellucida of the mammalian ovum; this historic observation was made on rabbit material. (2) Again using rabbits, he was the first to describe the cleavage of a mammalian zygote; he recognized that the products of cleavage are **cells;** and to the mulberry-like sphere of cells resulting from cleavage he gave the name in use today, the **morula.** (3) From his observations on cleavage, he was one of the first to reach the generalizations that all cells arise from pre-existing cells, and that in this process the nucleus plays an important part. Thus he was a pioneer in the study of cell division, and of Embryology in terms of cells. More than this summary can indicate, and in spite of his having no teaching appointment, Barry's personal example and friendships, especially with John Goodsir, exerted a powerful influence in the right direction during the early microscopic phase of the Golden Age of Anatomy in Edinburgh.

Allen Thomson (1809-84) graduated M.D. (Edin.) in 1830, his thesis being 'On the Development of the Heart and Blood Vessels in Vertebrate Animals'; thus he was an embryologist from the start. Then came a medical tour of Europe; and from 1831 to 1836 he taught Physiology extramurally, in association with the anatomical teaching of Sharpey. Intending to become an anatomist, Thomson also assisted Sharpey in Anatomy. After

an interval, during which he was successively physician to the Duke of Bedford, and Professor of Anatomy in Marischal College, Aberdeen, Thomson resumed the extra-mural teaching of Anatomy in Edinburgh in 1841. In 1842 he gave a very successful course of lectures on Microscopic Anatomy and Embryology, the first definitive teaching of Embryology in Edinburgh, and probably in Britain. In the same year he was appointed Professor of the Institutes of Medicine (i.e., Physiology) in the University of Edinburgh; six years later he became Professor of Anatomy in the University of Glasgow, a chair that he occupied with distinction for nearly thirty years. Though he made no great discovery or grand generalization, Thomson's description of two early human embryos in 1839 is a classic; and his influence in developing embryological teaching entitles him to be regarded as the father of Human Embryology in Britain. His 1842 course is a landmark in the history of the Golden Age of Anatomy in Edinburgh; and through him the brilliant light of that age penetrated to Glasgow.

Our mention of **John Hughes Bennett** (1812-75) must be brief. Following graduation as M.D. (Edin.) in 1837, Bennett travelled in Europe, where he came under the same influences as Sharpey, Barry and Thomson. Consequently, upon his return to Edinburgh in 1841 Bennett initiated a course in Histology, comprising both lectures and **practical work.** This was the year before Allen Thomson's lecture course. Bennett also taught Pathology, and used the microscope in that subject. Thus he introduced the regular use of the microscope into practical medical teaching in Edinburgh. Of his subsequent distinguished work as a physician this is not the occasion to speak.

John Goodsir (1814-67)

We now approach the greatest of all Edinburgh anatomists, John Goodsir. Born of medical ancestors in the village of Anstruther in Fife, in 1814, Goodsir was apprenticed to Robert Nasmyth, then the leading dentist in Edinburgh (whose name is perpetuated in Nasmyth's membrane), while he attended medical classes in Edinburgh. He studied Anatomy under Knox, who inspired him with lifelong devotion to that subject; he studied Surgery under Syme, then an extra-mural teacher; and in 1835 he obtained the medical licence of the Royal College of Surgeons. The next five years were spent assisting his father in practice in Anstruther. There the seed sown by Knox began to sprout. The marine fauna of the Firth of Forth furnished material for private investigations in Zoology and Comparative Anatomy that determined his bent toward that aspect of Anatomy for which the German writer Goethe had coined the term Morphology. The influence of his dental master is seen in a paper 'On the Origin and Development of the Pulps and Sacs of Human Teeth' published in 1839; this at once gave him a reputation in the young science of Embryology. This was also the year of Allen Thomson's reputation-making paper on early human embryos, of Barry's first Royal Society memoir, and of Schwann's **Mikroskopische Untersuchungen** on the cell. It was indeed a Golden Age.

In 1840 Goodsir removed to Edinburgh and engaged in private research. The next year he was appointed Conservator of the Museum of the College of Surgeons, giving lectures on the museum specimens and on his microscopic researches. From 1843 to 1845 Goodsir became successively Curator of part of the University anatomical museum, demonstrator of Anatomy (under Monro III), and finally Curator of the entire museum. He was succeeded as Conservator of the College of Surgeons Museum by his younger brother, Harry, who perished with the ill-fated Franklin Expedition in 1845.

In 1846 Monro III retired, and Goodsir applied for the chair. By this time he had a considerable reputation, based upon nearly thirty research papers, the chief of which (together with a few by his brother) had been published in 1845 as a volume entitled **Anatomical and Pathological Observations.** But some of Sharpey's friends sought to persuade Goodsir to withdraw in favour of Sharpey. Goodsir roundly declared that he would yield to no anatomist in Britain save only Richard Owen, and would stand or fall on his merits. But neither Sharpey nor Owen became candidates, and on his merits Goodsir was elected, in the year after Knox's departure from Edinburgh. One of the candidates was Wharton Jones. Goodsir was the first full-time Professor of Anatomy in the University of Edinburgh.

Goodsir's first task was to modernize his department, especially in its teaching; to the latter he devoted so much attention that his research diminished. Not that he lost interest in it, but his ability to bring his numerous enquiries to fruition declined, a situation aggravated by his failing health, and culminating in the tragic welter of investigations left unfinished, or merely planned, at the time of his death in 1867, after twenty-one years as Professor. In his later years he suffered from severe ataxia, and **post mortem** considerable atrophy of the spinal cord was found.

The two volumes of Goodsir's **Anatomical Memoirs** (published posthumously under the editorship of his successor, William Turner) are among my most treasured possessions, together with an engraving of Goodsir that belonged to Turner, who was Principal of the University of Edinburgh in my student days. Any attempt to summarize Goodsir's researches here is out of

the question, but one or two may be mentioned.

As we have seen, the cell doctrine 'caught on' in 1839, and for the next year or two Goodsir devoted himself partly to problems of the cell. I cannot do better than quote McKendrick's statement of Goodsir's position in this regard.

> 'In 1841 Henle showed that cells may multiply by budding, and in the same year Martin Barry observed that the reproduction of cells was accompanied by division of the nucleus. In 1845 Goodsir first promulgated the doctrine that cells never originate without pre-existing cells, a doctrine subsequently adopted by Remak and applied to pathological phenomena by Virchow.'

In his 'Observations on the Structure and Some of the Pathological Changes of the Kidney and Liver,' published in 1842, Goodsir wrote:

> 'I conclude that certain of the diseases of those organs are due to the development of new cells and new matter within the ducts and nucleated cells of the organs, in accordance with the normal laws of cellular development.'

It is not easy for us, accustomed as we are to trying to explain the abnormal in terms of the normal, to realize how revolutionary was this idea, that the bodily mechanisms underlying both health and disease are the same. This was also the view of Henle, and it was championed by Virchow. I cannot forbear quoting the dedication of Virchow's epoch-making **Lectures on Cellular Pathology** (1858):

> 'To John Goodsir, F.R.S., etc., Professor of Anatomy in the University of Edinburgh, as one of the earliest and most acute observers of cell-life, both physiological and pathological, this work on Cellular Pathology is dedicated as a slight testimony of his deep respect and sincere admiration by the author.'

Of all Goodsir's researches, almost the only ones referred to in the anatomical literature of today are those on the dynamic anatomy of joints. These are quite in the tradition of the Bells, but with geometrical refinements that would doubtless have puzzled those worthy brothers. Let me illustrate from his paper 'On the curvature and movements of the acting facets of articular surfaces.'

> 'The equiangular spiral, in its more general form as a curve of double curvature, is the only geometrical curve which fulfills the conditions of the successive movements and adaptations of articular curvature now under consideration. A characteristic property of this spiral, and one which peculiarly adapts it for generating the curvature of the surfaces of organic joints, is the geometrical similarity of all portions of any given example of curve which subtends the same polar angle, however different their linear dimensions may be; so that if the spiral be conceived as revolving round its pole, in the plane of two lines diverging from the pole, the lines will intercept an infinite number of geometrically-similar portions of the curve, but which become infinitely smaller or greater as the curve advances in the direction of its pole or away from it.'

Could anything in its field sound more modern? Anyone who wonders why Goodsir was considered a great anatomist should read his lectures 'On the employment of mathematical modes of investigation in the determination of organic forms,' delivered in 1849. Only today is his mathematical lead being followed, though the leader is largely forgotten.

As might be expected from the foregoing, Goodsir's teaching was characterized by both breadth and depth—that was possible in those days. He saw to it that the teaching in his department was comprehensive, embracing the various aspects of Anatomy in the proportions that he considered sound. As with his former teacher, Knox, Goodsir's lectures were the outstanding feature of his teaching. The following account has come down to us:

> 'In his Lectures on Human and Comparative Anatomy he did not satisfy himself with giving a mere descriptive account of the various structures he was called on to expound, but he pointed out the relations of his science to physiology, pathology, histology, morphology, and development. It was to this mode of illustrating the dry details of anatomy, more perhaps than to any special faculty for exposition, that his success as a teacher was due.'

Goodsir's lectures were sometimes too profound and erudite for his hearers; yet they commanded respect, and had a stimulating quality attested to by accounts reaching us from enthusiastic students who regarded him with affection and admiration as a masterly teacher. Even today we can sense the broad sweep of his knowledge as we read those of his lectures that were printed: 'On life and organisation,' 'On the dignity of the human body,' 'On the morphological constitution of the skeleton of the vertebrate head,' and others. John Goodsir was indeed the Master Anatomist.

Conclusion

I might have mentioned others, especially in the Goodsirian phase of the Golden Age. There was John Struthers; there was Joseph Lister, who, though not an anatomist, profoundly affected the subject; and there was William Turner. Why do I speak of the Golden Age as ending with the

death of Goodsir? That is, of course, an artificial historical landmark, for a change was beginning to come over Edinburgh Anatomy for some years previously. As early as 1853 Goodsir's health began to fail, and when he needed a full-time demonstrator to share his burden of teaching, he had to import one from London—William Turner. The glory had departed. Sir William Turner was an anatomist of great distinction, and he and his successors (Cunningham, Robinson and Brash) have trained a unique series of anatomical professors, no less than five of whom have occupied our own Chair of Anatomy. But neither before nor since has Edinburgh seen such a constellation of anatomical brilliance as during, say, the quarter of a century from 1825 to 1850, when she could boast of Robert Knox, John Reid, Wharton Jones, William Sharpey, Martin Barry, Allen Thomson, and John Goodsir. Here, in the words of Sir Arthur Keith, 'we come face to face with one of the most wonderful groups of young investigators ever produced by Edinburgh, or by any other capital of Europe.'

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Victorian Order of Nurses

The Annual Meeting of the Victorian Order of Nurses was held in January 1954. It was reported that our nurses made 34,393 visits to patients in the Greater Winnipeg area. The nurse travels to the patient's own home and there she remains long enough to carry out the attending physician's orders. The average length of her visit is nearly one hour—this includes travel time.

How do patients come under our care? The majority are referred by their own physician who telephones directly to the Victorian Order of Nurses' office giving instructions regarding the care of his patients. This is most satisfactory. Any family may request Victorian Order care, but since no care is given except under the doctor's direction, we immediately phone him for orders. Other sources of referral are Hospital Out-Patients Departments where the orders are usually given by telephone and are confirmed in writing, with the doctor's signature, on a form suitable for the purpose. With increasing frequency the nurse on the hospital ward telephones doctors orders re patients being discharged from hospital but still requiring nursing care. The City Health Department, the Provincial Health Department, the Manitoba Cancer Research and Relief Institute and other community agencies also refer patients in need of nursing care.

During the 1953 epidemic of poliomyelitis some doctors believed that certain patients could be cared for at home providing someone there could be given instructions regarding their care. The services of our nurses were available and fifty-one (51) families received this help.

Among other patients cared for were those suffering from heart disease, arthritis and rheumatism, diseases of the central nervous system, cancer, and of course many mothers and newborn babies.

Several industrial plants have bought nursing service on a part time basis.

The Department of Veterans' Affairs has for some years had a contract with the Victorian Order of Nurses for provision of nursing care to certain veterans. Increased use of this service was made during 1953.

The Director of Nursing in concluding her report for the year made the following quotation:

"The Victorian Order of Nurses' aim is to leave the family a stronger and more united group, better able to cope with future health problems as a result of her service to them. She recognizes and respects their own capacity to bring about a return to health for the patient and the family unit, both of which are affected by the illness of one member. She sees her role as one of encouraging the patient and those who are close to him to use the resources that lie within themselves to promote healing. A nurse can do no more, for fundamentally these are the forces which heal the sick and maintain a wholesome life. Her bed baths, back rubs, hypodermic injections and other treatments are aids to this end. The support she is able to convey to the patient and family while she is giving care, helps them find their own powers to overcome disease and adjust to a permanent disability.

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Editorial

J. C. Hossack, M.D., C.M. (Man.), Editor

Therapeutics

Some months ago when Professor Illingworth was here he spoke on peptic ulcer among other things. One of the most important points he mentioned was the team work of his group. His colleagues are operating internists, equally qualified to treat that condition and others either medically or surgically as the occasion requires; and when a change is made it is the same men who make it.

This close association of workers, it was pointed out, is of immense value in the conduct of a case and might be extended to other fields. So many special skills are necessary for the proper care of a patient that close team work is essential for best results.

I was reminded that about two years ago Professor Illingworth addressed a post-graduate gathering in the University of Aberdeen upon the topic "Peptic Ulcer." I did not attend, but the following morning the newspaper devoted over a column of its meagre front page to a summary of what he said. The caption was "Baking Soda is Best for Ulcer." So far as medical treatment went, the article (and Professor Illingworth) said, baking soda had yet to be improved upon.

After his address here I mentioned this incident to him and he assured me that he had not changed his mind. "Soda bic" had no solid contraindication and he still employed it. As this coincided with my own opinion I said: here is one who speaks with authority and not as the scribes.

And, indeed, he did speak with authority because, a year before, he had had to cancel a lecture on peptic ulcer—he was having his own removed!

The recent revival of chloral hydrate shows how dependent we are upon the manufacturing chemists for therapeutic guidance. Was there ever a time when chloral was **not** good when indicated? Was there not a time when its use was wide? But it was not advertised and so fell into almost complete desuetude.

Phenobarb took its place and very quickly the public came to recognize every small, white tablet as "a phenobarb." This made it difficult to give different members of a family the impression that they were being given different medicines. The chemists, however, came to our aid, and now a "phenobarb" can be prescribed in every colour of the rainbow and in a variety of shapes. Thus five separate members of the same family can all be taking the same drug and be ignorant of the fact. To such miserable trickery are we forced to stoop because of the layman's increase in pharmaceutical knowledge.

There is, however, one preparation that stands alone in its class—"Tabloid" Nitroglycerine. The "Tabloid" has a firm base that will resist weeks of carrying around in a pocket. It does not crumble. It needs no box or bottle to protect it. Therefore the sufferer from angina is never exposed to the embarrassment of having to extract a box from his pocket and a tablet from the box in view of curious onlookers. Too often the pain is tolerated so that the embarrassment may be spared. But with "Tabloid" the hand passes unseen from pocket to mouth and the anguish stayed as soon as it has started. Why this supreme advantage of the Tabloid preparation over all its competitors has not been exploited, I do not understand.

Doctors follow the fashion of the moment almost as slavishly as do women. Much of the average practitioner's up-to-date knowledge comes from advertising literature. The matter is reduced to a simple, easily followed summary which is read and absorbed more easily and more quickly than would be the original articles. The advantages of the particular brand advertised are stressed and believed. So do new remedies come into common use.

To aid their clientele still further the chemists send members of their faculties (the detail men) to give brief, scientific lectures and so between filling the ear and the eye these gentlemen keep us abreast of the times. It might be interesting to have them go one step further—to put on little courses starting with a dinner and consisting of a programme in which the various representatives would give illustrated lectures on their wares to which the audience, glass in hand, could listen or not as they wished.

Obituary

George Edwin Bruce

Dr. George Edwin Bruce died on January 30 at his home in Swan River, Manitoba, after forty-six years of service in his community. Born in Barrie, Ontario, he came to Winnipeg with his parents as a boy, and obtained his degree in Arts from Manitoba College in 1894. After teaching in Treherne, Emerson and Strathclair, he graduated from Manitoba Medical College in 1901 and began practice at Swan River. In 1947 he retired, but continued to take an interest in golf and curling. He is survived by his widow, a son and a daughter.

Ward Rounds

Children's Hospital, Winnipeg

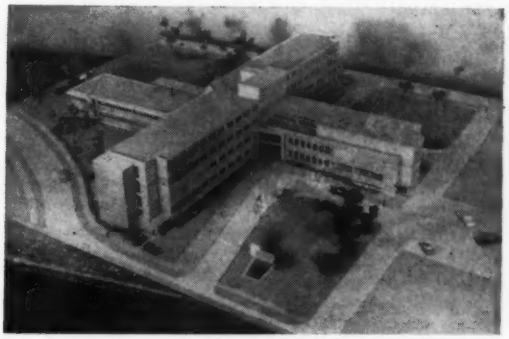
Edited by Wallace Grant, M.D.

Portal Hypertension

Case presented at Ward Rounds on January 7th, 1954.

Case No. 53-3693. This eleven and one-half year old boy was admitted on December 27th, 1953, because twice during the past year he had had episodes of vomiting gross blood, for which he had been hospitalized, investigated and treated in Moose Jaw. In January of 1953, although previously seemingly in good health, he suddenly developed an illness with fever, headache and a sore stomach. The following day early in the morning he reports that he had "a terrible feeling in my stomach", followed almost immediately by vomiting of dark material, which looked like blood. There was one other episode of hematemesis that night and during the following day he was quite weak. The next day he was admitted to Moose Jaw hospital, remaining there five weeks during which time he received four transfusions and a complete investigation for bleeding tendency. He was discharged on a high protein diet and remained quite well until about November 23rd when he began to have a heavy cold associated with pain at the top of his head. That same afternoon he suddenly felt dizzy and weak and had a temperature of 101 degrees. The following morning he vomited what appeared to be clots and dark granular material so that he was again admitted to hospital. At that time he had a hemoglobin of about 25 per cent and during this hospital stay he again had seven or eight transfusions. Investigation at that time tended to support a diagnosis of portal hypertension, hence he was referred here for possible surgical treatment. Apparently at the time of the first bleeding episode, he had melena but no fresh blood in his stools. He has at no time had any definite swallowing difficulty although he does occasionally complain of pain in his neck. He has always bruised easily, (and at the time of admission had a large bruise on his leg,) but other than this, and the gastro-intestinal bleeding, there has been no other abnormal bleeding tendency.

There were no abnormalities noted at birth, or in the immediate neonatal period, but he had what is reported as an abscess in the knee joint at the age of three weeks. His surgical history has included a right inguinal hernia repair in 1945, an appendectomy in 1949, and a broken right tibia which was treated with a Vitallium plate in 1949. There was apparently no unusual hemorrhage at the time of the operations in 1949.



On admission here, the boy was apparently in good health, in no distress and of fairly good colour. The apex impulse was maximal in the fourth interspace inside the mid clavicular line, the blood pressure was 115 over 65, the pulse rate was 90 regular, and there was a soft systolic murmur. The liver edge was felt one finger below the costal margin. The spleen could be felt to a level almost three fingers below the costal margin of the left, and it was very firm. Scars from the previous appendectomy and herniotomy were the only other abnormalities of the abdominal examination. Rectal examination was negative, although the stool was almost black in color (possibly due to prolonged iron therapy.) The only abnormalities about the limbs were several small old bruises, and one new one on the back of the right calf, and there was a linear scar over the right tibia.

Investigation showed no abnormality of red cells, white cells or prothrombin time, the hemoglobin was 12.2 grams per 100 c.c., and the platelet count was 54,000. Liver function tests were: serum-proteins, 7.2 grams per 100 c.c., Thymol turbidity 3 units, thymol flocculation 4+, cephalin cholesterol flocculation 3+.

X-ray studies were reported on by Dr. Childe as follows: "Esophagus, stomach and duodenum — The presence of a few small varices in the distal portion of the esophagus is quite possible, but the remainder of the esophagus is not abnormal in appearance. No abnormality of the stomach or of the duodenum is shown. Intravenous pyelogram— There is a good deal of barium in the colon. The spleen is considerably enlarged. Both kidneys function properly, and no definite abnormality of the urinary tract is seen. It is partially obscured by the barium. The left kidney is slightly lower than the right, presumably due to the enlargement of the spleen." Following this investigation the boy was operated on on December 31st, at which time Dr. Ferguson performed a splenectomy and a spleno-renal vein anastomosis.

Dr. Ferguson: Briefly, this boy had two massive gastric hemorrhages with vomiting of blood. With this history and the large spleen, presum-

ably he had portal hypertension, with bleeding from esophageal varices. Liver function tests suggest some mild liver disorder. The depression of the platelets and a slight depression of his leukocytes (6,300) suggest a mild degree of hypersplenism. I felt he should have a splenectomy and a spleno-renal anastomosis in order to decompress his portal system. He was operated on on December 31st, using a thoraco-abdominal approach with very wide exposure. The spleen was found to be about two and one-half times its normal size, it had a fibrotic capsule which was grayish and thicker than usual, and the spleen was quite firm. There was evidence of an extensive collateral circulation, coursing up over the spleen through the gastro-splenic ligament, over the stomach and around the esophagus. The veins in the portal distribution were distended. Because of technical difficulties we were unable to obtain portal venous pressure, but there was good evidence that it was increased. His spleen was removed. He had a good splenic vein which was turned down and the end was anastomosed into the side of his left renal vein. When this was completed the splenic vein seemed kinked but following some manipulation of the tail of the pancreas, the shunt from splenic vein through renal vein to inferior vena cava looked as if it would be satisfactory. Post-operatively the child has done well. His platelet count has now risen to 264,000. If the splenic vein anastomosis stays open, he should have no further episodes of esophageal hemorrhage.

Dr. Hoogstraten: The gross appearance of the small wedge of liver removed at the operation, and the spleen was normal. Microscopically the liver shows normal histology and cytology. The parenchymal cells stain well and normally. There is no increase in fibrous tissue in the portal tracts, no evidence of cholestasis, and no inflammatory infiltrate. The capsule and fibrous trabeculae of the spleen are moderately thickened, and there is no apparent increase in fibrous tissue in the sinusoidal walls. The Malpighian bodies are normal and the pulp contains a normal cell population. There is no evidence of extramedullary haemopoiesis. The anatomic diagnosis is, normal liver and congestive splenomegaly.

Dr. Ferguson: Portal hypertension may manifest itself in two major ways, either with signs and symptoms due mainly to portal obstruction, or with the clinical manifestations of hypersplenism. Hypersplenism may dominate, with depression of platelets and leukocytes, without significant portal obstruction and esophageal bleeding. This boy had a combination of both. The obstruction can be in the liver, for instance with cirrhosis, or it can be extrahepatic with a cavernous type of transformation in the portal vein or it can be due to a congenital valve. The clinical picture then can be esophageal hemorrhage, or anemia and patechial hemorrhages due to the thrombocytopenia of hypersplenism. If liver disease predominates there may actually be hepatic failure.

Of the eleven patients with whom I have had some experience who have had a spleno-renal anastomosis, all but one have had a good result without further bleeding. In some instances the operation was performed less than six months ago so that the follow-up cannot be considered significant as yet. One child was two and one-half years old at the time of his operation. His splenic vein was very small, and six months later he had another episode of hematemesis. When he was re-explored, the splenic vein was found to be thrombosed. Unfortunately his obstruction was in the portal vein, so it was impossible to do a porto-caval anastomosis. When he has further hemorrages, all that can be done now is a local resection of the veins in the esophagus, which can give only temporary relief. All the other children have been older, with splenic veins of adequate size and so far they have done well. One of these children had shown progressive jaundice after birth and at the age of three months a diagnosis of biliary atresia was made. The child was operated on by Dr. William Ladd who found an atresia of the common duct, but with a patent duct system above, so that it was possible to do an anastomosis of the patent portion of the common duct to the duodenum. Following this operation the child had several bouts of cholangitis, but subsequently did well. At the age of twelve years she came back into the hospital with an enlarged spleen, some depression of her leukocytes and platelets and slightly abnormal liver function studies. Esophageal varices were demonstrated radiologically although she had not had any hematemesis. Her spleno-renal anastomosis was performed last June, and when I last heard of her in July she was doing well.

Dr. Medovy: Was there not a simpler procedure advocated a couple of years ago by Reinhoff of Baltimore which involved tying off the gastric artery just beyond the point where the gastro duodenal artery came off? Is this procedure still held in good repute?

Dr. Ferguson: He tied off the hepatic artery, which is certainly a simple operation to perform. Since then there have been reports in the literature of hepatic coma following the ligation and the procedure is being abandoned for this reason and also because the patients bleed again.

Dr. Chown: What was the rationale of tying off the hepatic artery?

Dr. Ferguson: As I understand it, when the liver is cirrhotic, the portal vein flow is obstructed so that very little venous blood is going through the liver. In compensation for this, an excess of blood goes through the hepatic artery and Reinhoff and his associates feel that there is a reversal

of flow so that hepatic artery blood goes into the sinusoids, has difficulty making its way to the hepatic vein, and backs up into the portal system. By reducing blood supply, they hoped to reduce the portal pressure.

Dr. S. Israels: I believe Johnson had an article some years ago in the Canadian Medical Journal in which he described the method he used to determine whether or not these shunts are working. Have you had any experience with this, and what is the principle of the method?

Dr. Ferguson: They catheterize the inferior vena cava and get the tip of the catheter just opposite the point where the left renal vein is emptying. The child is given oral glucose, following which they take in rapid succession, a series of blood glucose determinations from the arm vein and from samples taken through inferior vena cava catheter. They feel that if the spleno-renal anastomosis is working, blood glucose should become elevated sooner in the inferior vena cava than in the peripheral circulation. It is a rather complicated test, which so far as I know was done on only one patient, and I don't know how accurate it is.

Dr. S. Israels: If the X-ray examination of the esophagus was not too effective in demonstrating varices, how can you be sure that this was the site of the bleeding and that he did not have a peptic ulcer?

Dr. Ferguson: No, we examined the stomach and duodenum by X-ray, and there was no ulcer apparent and at the time of operation we could not feel any evidence of ulcer in stomach or duodenum. Besides this, peptic ulcer is not usually accompanied by the large spleen, damaged liver, evidence of hypersplenism, and the definite collateral circulation that was apparent at the time of the operation.

Dr. S. Israels: Still, if you could not be certain of the site of the bleeding, should he not have been esophagoscoped?

Dr. Ferguson: Esophagoscopy is not without risk in these patients. It can produce a massive hemorrhage.

Dr. Lionel Israels: What do you feel is the underlying pathology in this instance?

Dr. Ferguson: I am not satisfied that we know. There is no history of infectious hepatitis or other liver disease, peritonitis or intra-abdominal abscess and there was no neonatal periumbilical infection. He did have his appendix removed and I thought that perhaps the appendicitis might have been followed by a pylephlebitis with extension up into the portal vein. However, at that time he had had pain for only about eight hours, and the condition was described by the surgeon as "early appendicitis", he had no fever or chills, and was home on the fourth day.

Dr. Desmarais: What is the possibility that this could have been Gaucher's Disease?

Dr. Ferguson: The pathological report on the liver biopsy and the spleen would rule out Gaucher's disease. He did have a bone marrow examination at one time, and no Gaucher's cells were seen. However, as far as I know, Gaucher's disease does not produce esophageal varices with gastro intestinal hemorrhages. Its main clinical feature is the hugely enlarged spleen and evidence of hypersplenism.

You might be interested in the boy L. K. who was presented here about a month ago as a possible candidate for spleno-renal anastomosis. He had had a splenic artery ligation as an emergency procedure because of recurrent massive hemorrhages some months before. At the time of operation we found no splenic vein so could not do a spleno-renal anastomosis, but had to be content with a splenectomy alone. After the operation he had a very stormy course and a massive gastro-intestinal hemorrhage, but he did recover and it is now a month since his operation. He is at home, has good colour, and has gained three pounds. He may just happen to be an example of that rare form of portal hypertension which can be cured by splenectomy alone, the block being primarily in the splenic vein, or in the spleen itself.

Dr. Grant: He would then have been cured after the splenic artery ligation if this were so, wouldn't he?

Dr. Ferguson: You would think so, but these children have commonly such an extensive collateral circulation that portal hypertension can persist even after a splenic artery ligation.

Dr. Ross: Has anyone been injecting these spleens, following removal, with plastic materials, which make a cast of the internal structure?

Dr. Ferguson: No, this would probably not be of too much help, but sometimes before the operation we inject the spleen with diodrast through the abdominal wall, and take rapid serial X-rays in order to follow the radiopaque material through the portal system. We didn't do it in this boy, but we did do it on L. K. and no diodrast appeared in the portal system. Instead it flowed up over his stomach around the esophageal varices. I thought this was probably due to technical error on my part, that perhaps I had not put the needle in the right place since it is a blind manoeuvre. At operation the spleno-portagram was proven to have been correct,—there was no functioning splenic vein.

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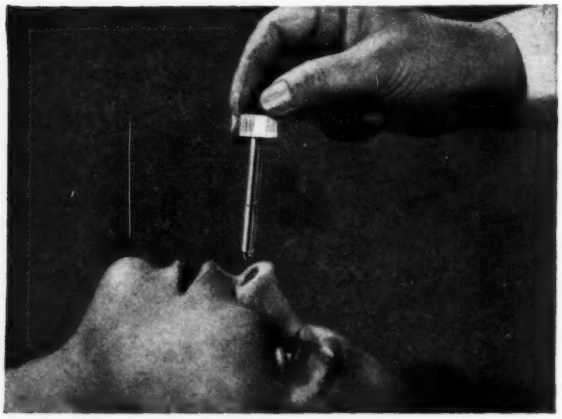

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Article

The Phases of Alcoholism*

The idea and the possible importance of phases in the course of alcoholism were first formulated by E. M. Jellinek in 1946 on the basis of a questionnaire study of 100 members of Alcoholics Anonymous. More recently the same investigator has elaborated his concept of phases after analyzing the returns from a larger questionnaire administered to more than 2,000 male alcoholics. From the point of view of those who deal in a professional capacity with both addictive and non-addictive alcoholics, the ability to pigeonhole the patient with respect to which stage of the illness he has reached might prove helpful in planning a programme of treatment.

All use of alcoholic beverages begins with a social motive, and even the prospective addict may show little or no deviation from ordinary social drinking in the early stages of his drinking pattern. In contrast to the average social drinker, however, the future alcoholic feels what Jellinek describes as "a rewarding relief in the drinking situation. The relief is strongly marked in his case because either his tensions are much greater than in other members of his social circle, or he has not learned to handle those tensions as others do." This is the beginning of the "pre-alcoholic" phase, the first of the four phases outlined by Jellinek. In this phase the alcoholic may for a time be quite unaware of his peculiar psychological reaction to alcohol. He may seek relief in intoxication only rarely.

Just a Little More

Some alcoholics never go beyond the first stage. The prospective addict, however, will learn to depend more and more on this method of relieving tension and, in the course of a few months or years, he will come to indulge in relief drinking almost daily. He may still not be drinking with the conscious intention of getting drunk. Nevertheless the degree of sedation which he seeks requires fairly large amounts of alcohol. It is during this phase of constant relief drinking that the individual may find that he must drink a little more than formerly in order to attain the same surcease from emotional stress.

"Blank-outs" May Be the First Warning

The pre-alcoholic phase gives way to the second or "prodromal" phase at the time the alcoholic experiences habitual "blackouts" after medium as well as intoxicating amounts of alcohol. These amnesias are really "blank-outs," for there is no loss of consciousness as in true "blackouts." Rather, the individual may perform a series of ordinary or unusual actions during a drinking episode but afterward he will have no memory of what he did. Even the normal social drinker may experience this type of amnesia on some occasion when he drinks enough while in a state of exhaustion. It is the frequency of the "blank-outs" and their occurrence after drinking only medium amounts which should warn the alcoholic or his therapist that the addictive phase of alcoholism may be close at hand. At about this time, too, certain physical changes may be seen in the drinking behavior of the individual who is experiencing "blank-outs." Secret drinking, thinking about alcohol much of the time, the development of guilt feelings about drinking, and deliberate avoidance of reference to alcohol in conversation, are common symptoms of this prodromal phase.

Sequence and Type of Symptoms Vary

Although not all the symptoms occur in all alcoholics, and they do not necessarily follow the same sequence, the phases and the order of symptoms within each phase proved to be characteristic for most of the 2,000 alcoholics in this survey. They can thus be thought of as representing an average trend among men alcoholics.

The prodromal phase may last from 6 months to 4 or 5 years, depending on the physical, psychological and social status of the individual. The drinking at this stage, although not yet conspicuous, has gone well beyond ordinary usage and may be starting to interfere with the metabolic and nervous health. Since the alcoholic at this point is capable of some insight into this growing demand for alcohol and may feel acute fears of where it is leading, this becomes an important moment at which the developing addiction may be intercepted. In recent years the publicity given to the symptoms of alcoholism has caused many alcoholics in this phase to seek medical help. Some alcoholics remain at this non-addictive phase indefinitely.

The prodromal phase ends and the "crucial" or "acute" phase begins with the onset of loss of control, which is the critical symptom of addiction proper. This symptom means that any drinking of alcohol may start "a chain reaction which is felt by the drinker as a physical demand for alcohol." This feeling may take hours or weeks for its full development on any occasion; it lasts usually until the alcoholic is too intoxicated or too sick to drink any more. After recovery from the episode, it is not a physical demand which leads to the next bout, but rather the original emotional tensions or even a social occasion involving drinking.

*The Committee on Alcoholism for Manitoba, Copyright by Journal of Studies on Alcoholism, Inc., Yale University, New Haven, Conn.

Drinking Behavior Rationalized

The alcoholic in this phase can still go through periods of voluntary abstinence and exert control in other ways, but once he starts to drink he cannot be sure he will be able to stop. At this stage he is relatively difficult to treat, because he has not yet admitted that he has undergone a process which makes it impossible for him to control his alcohol intake and he still believes that it is a matter of will power which he can and must master in the future.

Other new symptoms begin to appear now. The alcoholic begins to rationalize his drinking behavior, finding good reasons to justify every episode of intoxication as well as other trouble-making actions. Since by this time the drinking behavior has become conspicuous and painful to all concerned, the addict is now subjected to strong social pressures and warnings. These too are met by rationalizing by convincing himself that the fault lies not with him but in others. The addict now begins to show marked aggressive behavior. He tries to make up for his inner feeling of failure by acting like a "big shot" and "big spender."

The Pangs of Remorse

Even in the prodromal period, remorse over drinking was felt sometimes; but now the alcoholic is constantly troubled by remorse and the only way he knows to meet this is by further drinking. In a desperate effort to regain control, he may try such measures as changing the pattern of his drinking—switching to beer, perhaps—or even "going on the water-wagon" for short periods, or trying the "geographic cure" by moving to a new town. When he detects that some friends are getting ready to drop him or that an employer is about to dismiss him, he may "beat them to the draw" by dropping the friends or quitting his job.

His entire behavior grows steadily more alcohol-centred, and he continues to drop all interests except drinking; he interprets all his relations with people in the light of his need for drink. He worries a good deal about his supply of alcohol and will try to keep a stock hidden away. Neglect of nutrition aggravates the effects of excessive drinking and at this point he may have to be hospitalized for the first time. His sexual activity may decline and at the same time he may show exceptional jealousy toward his wife. As his life becomes utterly disorganized he may resort to regular morning drinking although previously he had done this only at times. This behavior marks the end of the crucial phase and ushers in the last or "chronic" phase.

Bouts of Drunkenness

During the crucial phase the alcohol addict has fought against complete loss of social foot-

ing. He has tried, for example, to avoid daytime intoxication, and has managed to keep up some sort of front. But the process which started with the onset of loss of control over drinking has progressively undermined his resistance, and the struggle against the "demand" set up by morning drinking eventually proves too great. He now succumbs to long bouts of drunkenness, the major symptom which initiates the chronic phase of alcohol addiction. At the same time he more or less gives up the struggle against alcohol. The symptoms which accompany the phase of addiction vary between individuals. There may be severe ethical deterioration or impairment of thinking; 10 per cent of all alcoholics develop true phychoses. The addict in this phase will drink with persons far below his social level and will drink bay rum or rubbing alcohol if he cannot get regular beverages.

At this time the alcoholic may find himself getting drunk on less alcohol than formerly. Vague fears trouble him. His fingers may show a constant tremor, preventing the doing of simple acts unless he first has a drink or two to "steady" him. The need to control these symptoms caused by heavy drinking has now become greater than the need to relieve the original underlying symptoms which led to the alcoholism, so that the drinking takes on an obsessive character.

At this point the entire rationalization system fails and the alcoholic admits his defeat. A majority experience vague religious desires. Although the obsessive drinking continues, the alcoholic is now spontaneously open to treatment.

Addiction Can Be Arrested

Formerly it was thought that this stage of defeat must be reached before the alcoholic could be successfully treated. Clinical experience during the last decade, however, has shown that this is not the case. The course of addiction may be arrested at any point if an artificial "defeat" can be induced in the individual by the therapist. As knowledge of the disease process of alcoholism spreads, more and more incipient addicts voluntarily present themselves for treatment. Since the prospective addict can be sifted out from other types of excessive drinkers by a thorough examination of their symptoms, Jellinek believes that it may yet become possible to handle the problem of addiction at the preventive level.

College of Physicians and Surgeons of Manitoba

Business Arising from Minutes of Executive Committee — September 10, 1953

(a) Election of Council

The Registrar advised that at the May Council meeting it was agreed to leave the election of Council in abeyance until the Annual Meeting. He stated that normally the present Council would be in power until 1955. The communication from the solicitor was read, stating that there was no reason why the election should not proceed in the ordinary manner, since the statement in the amendment to the Medical Act "the first election of members of the council after the coming into force of this Act shall be held not later than one year following the coming into force of this Act, on a date to be fixed by the executive committee," is not mandatory.

Dr. Walton suggested that the election be held before 1955, since the amendments were assented to in April, 1953, and it would mean a gap of two years. He also suggested that the election not be held during the summer months when many of the members are absent from the Province on their holidays. According to the By-law, June 30th is the date on which the election lists are made up of members who are not in arrears of fees at that date, the nominating papers are mailed on August 1st and are returned by August 31st, and voting papers are mailed on September 15th and are returned by September 30th.

Motion: "THAT the election of Council be held in accordance with the By-laws, and the whole question of time of election be referred to the Legislative Committee for study." Carried.

(b) Amending of By-laws

The Chairman of the Legislative Committee reported that since most of the members of the Committee were from out of town, no formal meeting of the Committee had been held, but the matter had been discussed with the Registrar on several occasions. The amendments to the Medical Act were given Royal assent by the Legislature in April, 1953, and the By-laws were approved by Council on May 23, 1950. Since that date several notices of motion have been given, and minor changes made by the Executive and Council. According to the By-laws "No amendments or additions to any of the foregoing by-laws shall be made unless due notice setting forth the proposed amendments or additions shall have been given at a meeting previous to that at which the same comes up for discussion, and all resolutions of the Council inconsistent with the above by-laws are hereby repealed except as outlined under the powers of the Executive Committee."

The Registrar presented a letter from Dr. ....., which was received subsequent to the May meeting of Council, suggesting that the municipalities of Whitehead and Sifton might be included in the electoral district which now includes the municipalities of Cornwallis and Elton, since he is more closely associated with the members in Brandon, and is comparatively far removed from the majority of members in the southwest corner of the Province.

Notice of Motion: "THAT the By-laws, Rules and Regulations of the College of Physicians and Surgeons of Manitoba be consolidated in conformity with the 1953 amendments to the Medical Act, and changes recommended to-date by Council." Carried.

Motion: "THAT the problem of changing the By-laws be referred to the solicitor for his consideration." Carried.

Dr. Boyd stated that when legal opinion was received on the amended by-laws, they would be mimeographed and sent out to members of Council before they come to the meeting of Council in May.

(c) Registrars' Meeting

The Registrar reported that mimeographed copies of the minutes of the Registrars' Meeting, held in Winnipeg in June, 1953, had been circulated to members of Council, and that the luncheon had been paid for.

(d) Medical Registration Council, Dublin, Eire

The Registrar reported that Manitoba had reciprocity with Great Britain and Ireland until Ireland advised on April 4th, 1953, that they no longer had reciprocity with Manitoba with the passing of their new Act. The Registration Committee, and Council at their meeting in May, agreed that since Eire cancelled the reciprocity agreement they should reinstate it. A communication was received on June 5th, 1953, requesting that Manitoba Council make formal arrangement about reciprocity for registration, and that the Medical Registration Council of Dublin would ask the Government to make an order applying Section 26 of their principal Act to Manitoba.

Motion: "THAT no action be taken, and the Medical Registration Council, Dublin, Eire, be so advised." Carried.

(e) . . . .

(Refer Representatives to Medical Council of Canada).

(f) Auditors

(Refer Treasurer's and Auditors' Report).

(g) Purchase of Addressograph

(Refer Representatives to Liaison Committee).

Motion: "THAT the report of the Executive Committee be adopted, in view of the foregoing discussion." Carried.

B. Registration Committee

1. Since the last meeting of Council in May, 1953, the Registration Committee has met on five occasions.

2. Fourteen Enabling Certificates were granted and six applications for Enabling Certificates were deferred. Certificate of Registration was authorized in eighteen instances: Two were deferred and one was refused. Temporary Certifcates of Licence were granted to eleven applicants. Total number of registrations in Manitoba for the period 25th May, 1953, to 30th of September, 1953, was thirty-seven.

3. F/L ..... applied for registration under the Reciprocity Agreement with the General Medical Council of Great Britain. This Officer had qualified at the University of Aberdeen, 1950, M.B., Ch.B. He had no interneship and was commissioned under a short term service commission of three years with the R.C.A.F. Under our by-laws it was impossible to register him, but so that his duties with the R.C.A.F. would not be interfered with, he was granted a temporary certificate of licence and he was advised that when he had a year's interneship he would be registered in Manitoba without further qualification. Your Chairman communicated with the D.M.S. of the R.C.A.F. regarding the commissioning of Medical Officers without registerable qualifications: In reply, the Committee was advised that future appointments would be carefully scanned in this regard and the R.C.A.F. offered to extend F/L ..... commission to five years so that one of them could be taken as an interneship with full pay and allowances.

4. It is necessary again to report to Council on the matter of ..... During the month of August, a Persian Surgeon with outstanding British qualifications and by name, Dr. ....., while visiting in Winnipeg was kind enough to look over the documents of ..... The photostat which ..... submitted and which purported to be a Medical Degree from the University of Teheran was written in Parsee and we had never been able to obtain suitable translation of it. Dr. ..... translation indicated that the certificate was only one of a medical aide and not of a doctor. This translation was so alarming that a photostat copy was sent to the Consulate General of Iran in New York for a confirmatory translation. The Consulate General very kindly sent us a translation which, in effect, confirmed that of Dr. ..... Another photostat copy was sent to the Registrar of the Medical Council of Canada who submitted it to the Department of External Affairs, Ottawa. There, a translation was prepared by a Turkish woman who had some knowledge of the Iranian language. Her translation agreed, in a general way, with those mentioned above but was much less clear. In the light of this information, the Registration Committee advised your Executive that ..... Enabling Certificate should be cancelled and this action was concurred in by the Executive. The whole problem was placed before the Medical Council of Canada which concurred in our action. ..... was informed by Registered Mail of our action and a cheque was sent to him refunding the fee which he had paid for the Certificate.

The Registration Committee continues to be concerned with large numbers of applications from foreign doctors, both European and Asiatic. This province has been more generous in its handling of such applications than any of the other provinces and it would seem probable that the number of applications to this Province may increase in the years to come. Many provinces now require Canadian Citizenship for registration. Among these is the Province of Ontario. Ontario will grant an Enabling Certificate to a foreign applicant if he is otherwise qualified if he files a declaration of intention to become a Citizen of Canada, but the applicant is warned that he will not be registered in Ontario until he is a Citizen of Canada. As citizenship requires a minimum period of five years, it is evident that this province and others may expect to receive applications for registration from foreign doctors who have written and passed their Medical Council Examinations under an Enabling Certificate from another province such as Ontario, but had not yet been able to become Canadian Citizens. Citizenship is not required in the Prairie Provinces although there has been a move of this kind contemplated in Alberta. The Committee does not consider that Manitoba should require citizenship because of the obvious hardship which would ensue, but your Committee has been requiring a declaration of intention to become a citizen.

The Committee also has been concerned as to whether it should continue to entertain applications from foreign doctors who do not intend to practice Medicine in this province. Applications of this kind come largely from Chinese doctors and also from some American doctors, mostly colored, who plan to practise in the British Colonial Medical Service. Both of these categories come to Manitoba so that they may obtain Reciprocal British Registration. Your Committee has felt that in the present disturbed state of the world, that they should continue to grant assistance in this regard, but the Committee has become more and more concerned with the fear of abuse of the privilege. The Committee recommends to Council for its consideration a proposal that all foreign applicants should be required to appear in person before the Registration Committee.

The Committee also desires approval of Council for the printing of suitable application forms to

replace the present rather unsatisfactory mimeographed forms.

All of which is respectfully submitted.

C. H. A. Walton, M.D.,
Chairman.

16 October, 1953.

Motion: "THAT the report of the Registration Committee be adopted." Carried.

Dr. Williams stated that with regard to the granting to Chinese graduates of registration and Enabling Certificates to take the examinations of the Medical Council of Canada, he had once or twice expressed the opinion of disapproving of issuing them to graduates after 1948. After Communist control took over these schools, academic teaching was replaced by political indoctrination lectures, and the years and hours of study were reduced. He did not think that applicants who graduated after that time should be granted any certificates. Dr. Corrigan stated that Manitoba has reciprocity with Great Britain, and the Chinese graduates wish to return to Hong Kong, and the College of Medical Evangelists graduates wish to enter mission fields in British colonies. He said there was an element of sympathy and co-operation with the World Health Organization. Dr. Macfarland advised that all Chinese and European applicants were referred first to the Credentials Committee of the University of Manitoba for assessment of their basic education, and that the Chinese had postgraduate training in the United States as a rule.

Motion: "THAT the Registration Committee be instructed to require all foreign applicants to appear for a personal interview." Carried.

Motion: "THAT the Registration Committee be given permission to have suitable application forms printed." Carried.

To bring Council up-to-date on ....., the Registrar reported letters received from ....., dated September 25th; copy of letter addressed by ..... to his Winnipeg solicitor, dated September 26th; letter from ..... Montreal solicitor, dated October 1st; the Registrar's reply to ..... Montreal solicitor after consultation with our solicitor, dated October 13th; and letter from ..... dated October 15th, to which no reply has been sent.

C. Education Committee

The Chairman, Dr. A. R. Birt, reported no meeting of the Education Committee had been held during the year.

D. Finance Committee

Meeting held October 17th, 1953.

Present: Dr. C. E. Corrigan, Dr. B. Dyma, Dr. T. H. Williams.

After some discussion concerning investments it was moved by Dr. Corrigan, seconded by Dr. Dyma and passed "That the College of Physicians and Surgeons invest available surplus funds in the Current Account and Investment Trust Account bank balances up to a possible total of $6,000.00 by the purchase of Province of Manitoba 4¼% Bonds due October 1, 1968." Carried.

Moved by Dr. Dyma, Seconded by Dr. Corrigan "That the salary of Miss Lorna Zawadzki be increased to $165.00 per month effective from July 1, 1953." Carried.
and "That the salary of Miss Jean Allison be increased to $225.00 per month effective from July 1, 1953." Carried.

Moved by Dr. Dyma, seconded by Dr. Williams "That we approve Christmas bonus, $40.00 to Miss Allison and $25.00 to Miss Zawadzki, paid in 1952, and recommend the same for 1953.00 Carried.

Adjournment.

T. H. Williams, M.D., C.M.,
Chairman, Finance Committee.

Motion: "THAT the report of the Finance Committee be adopted." Carried.

E. Legislative Committee

The Chairman, ..., advised there was nothing further to report in addition to the discussion under Business Arising from Minutes of Executive Commitee.

F. Library Committee

Since your representative's last report in May of this year, there have been no meetings of the Library Committee to which I have been summoned. There is, therefore, nothing further to report.

T. H. Williams, M. D., C.M.,
Library Committee Representative
College of Physicians and Surgeons.

Dr. T. H. Williams presented the following statistics as prepared by Miss Ruth Monk, Medical Librarian:

Statistics

Contents of Library

1. Books, Bound and Unbound Serials (Periodicals): the approximate number of volumes in the Library, exclusive of the duplicate files of serials:

1952-53	1951-52
18,980 volumes	18,446 volumes

Progress

534 volumes, or 2.89% increase over 1951-52.

2. Serials (Periodicals): titles currently received:

	1952-53	1951-52
Titles	371	344
Duplicates	5	3
	376	347

Progress

An increase of 28 titles (all gifts but one), or 8.13% over the session 1951-52.

Volumes added to the Library by The College of Physicians and Surgeons' Grant: 135 Volumes.

This is an **increase of 24 volumes** from last year's purchases on this grant.

These 135 volumes comprise 37.81% of all purchases in 1952-53, and 27.43% of all total accessions.

Services Other Than Loans

A. References:

Reference Requests Made By:	Number	% of the Total Request
Winnipeg Physicians	75	34.40%
Rural Manitoba Physicians	25	11.47%

The doctors (including faculty) last year were advised of references to current journals for subjects of special interest to them. There were 76 persons so advised of 590 articles.

B. Book Lists:

The book lists of new additions to the Library were distributed to the profession, both in Winnipeg and rural Manitoba, who have NOT been USING the Library. The response from the rural doctors was most gratifying, as 17 of the 84 who received the lists, sent in for the material following its receipt.

Circulation Statistics — Borrowers and Loans — 1952-53

Class of Borrows	BORROWERS: Total Possible Borrowers	Actual Borrowers	% of Possible Borrowers	Increase or Decrease in Borrowers from 1951-52	LOANS: Total Items Loaned (Bks. & Jnls.)	Increase or Decrease from 1951-52	% Change from 1951-52
1. Registered Urban Physicians (Wpg. & Suburbs)							
a. Faculty	169	105	18.07%	Dec. 3			
b. Non-faculty	412	154	26.55%	Inc. 18 or 13.23%			
	581	259	44.58%		4,155	Inc. 577	16.12%
Increase in number of actual borrowers since 1951-52 — 15. Total number of items loaned to Winnipeg physicians — 4,155 items or 39.74% of ALL loans in 1952-53. This represents an increase of 577 items or 16.12% over 1951-52.							
2. Registered Rural Physicians	246	43	17.48%	Inc. 17 or 64.65%	262	Inc. 75	40.11%
TOTAL Registered Manitoba Physicians	827	302 (a)	36.51%	Inc. 32 or 11.85%	4,417 (b)	Inc. 625	17.31%

(a) An increase of 11 in actual members of registered physicians using the Library and the highest combined total on record (from 1921) for Winnipeg and rural Manitoba.

(b) Registered Manitoba physicians including licensed physicians on faculty borrowed 4,417 items of the total 10,456, or 42.24% of all loans in 1952-53.

October 6, 1953.

Motion: "THAT the report of the Library Committee be adopted." Carried.

Manitoba's Medical Men
4-Radio

The members of the Manitoba Medical Association have been gratified to see the number of subscribers to the Manitoba Medical Service increase year by year. However, they have noticed that the people in the rural areas have not participated to the extent anticipated. A few selected areas have been canvassed but the province is large and the task is great.

There are two reasons why the doctors are anxious to have a greater number of subscribers outside of the city. First, the greater the coverage, the greater the strength of the pre-paid medical plan and second, the treasurer of the Manitoba Medical Service indicated that since the cost per subscriber outside the cities is less than that in the cities, the cost per subscriber on a province-wide basis would be reduced.

In order to reach as many people as possible in the Province the Executive of the Manitoba Medical Association and the Board of the Manitoba Medical Service decided to use the radio to advertise the plan and at the same time to render a public service to the people of the Province.

A series of thirty-nine radio broadcasts was arranged to be given every Sunday afternoon at 4.45 p.m. over Radio Station CKY—580 on the dial. This station has a very long radius. The programs can be heard outside the borders of the Province East, West and South and up to the Pas to the North.

The programs have been approved by the American Medical Association and have been run in Calgary. The topics covered include such diseases as tonsillitis, anaemia, fractures, arthritis, diabetes and many others.

The first eight programs are sponsored by the Manitoba Medical Association and the subsequent ones will have a joint sponsorship by the Manitoba Medical Association and the Manitoba Medical Service, with the continuity planned to inform the public about the advantages of joining the doctors' pre-paid medical plan which we firmly believe is the best one in the world.

L. A. Sigurdson, Publicity Editor.

Manitoba Hospital Service Association

Significant Changes in Blue Cross Benefits

Prepared by Blue Cross for the guidance of the Medical Profession upon the suggestion of the Manitoba Medical Association

On January 1, 1954, new Blue Cross contracts became effective. Increased subscription charges largely reflect the need for increased payments to hospitals, but are in part due to the need to adjust benefits to present day needs.

Major Benefit Changes Include:

Days of Care:

21 days at full benefits during the year of first joining.

31 days at full benefits each calendar year thereafter.

10 days additional at full benefits (bonus days) if no care on the contract has been received the previous calendar year—or earlier within the year —the contract having been in force 12 clear months.

(Note: that admission for trivial reasons thus are disadvantageous to the subscriber in the event of a serious illness. This should help you in resisting requests for unnecessary hospitalization.)

Where full benefit days are all used, additional days at 50% benefits are provided up to a total of 120 days.

Days of care are calculated "per illness". They are provided for each unrelated illness or condition and the illness may overlap a calendar year. In determining whether an admission is related to a previous admission, an interval of three clear months between admission and prior discharge will be accepted as presumption of non-relation.

Anaesthesia:

Now that the anaesthetists are practicing their specialty on a fee-for-service basis and no longer as employees of the hospitals, the services of the anaesthetists can no longer properly be treated as hospital service and such services are not covered by Blue Cross Contracts. While you know that any possible saving to Blue Cross as a consequence has been absorbed by an increase in operating room charges, your patient may not understand this.

Still covered without limitation are anaesthesia material and use of equipment provided by the hospital.

Drug Benefit:

All drugs of therapeutic value are covered up to a maximum of $25.00 for each admission.

The Deductible Feature:

On all Community and Non-Group contracts, a $15.00 deductible is provided for. The patient pays the hospital the first $15.00 of the hospital's charges for the contract benefits.

On Semi-Private and Ward Group contracts, such deductible is not ordinarily required; the subscriber may, however, to obtain a lower subscription rate, choose such a deductible contract.

On Casualty cases and admissions for minor surgery, application or change of cast, the patient pays the hospital the first $5.00 of its charges under all contracts.

(Note: the deductible feature does not apply on either maternity or T. & A. admissions which are otherwise limited:)

Maternity:

The maternity benefit is $60.00 per pregnancy on Ward and Community contracts; $75.00 per pregnancy on Semi-Private contracts.

As previously, benefits as for any other illness are provided in cases of (a) Caesarian Section; (b) Ectopic pregnancy; (c) Eclampsia; (d) Premature termination of pregnancy without child-birth.

T. & A.:

2 days care is provided.

Mental and Nervous Conditions:

These are still excluded by all contracts, **except:** provision is made for benefits where active treatment is required as follows: 21 days care for electro-shock therapy, (maximum 105 days during the life of the contract.) 12 days care for sub-shock insulin therapy (maximum 60 days during the life of the contract.)

Diagnostic Admissions:

Admissions primarily for diagnosis or primarily for physical therapy are not covered, **except:**

Admissions primarily for diagnostic services which are not available otherwise than in a hospital are provided as follows: Air encephalogram, ventriculogram, myelogram, cerebral angiogram, angiocardiogram, electromyogram and diagnostic curettage; and if a general anaesthetic is administered, gastroscopy, bronchoscopy, cystoscopy and sigmoidoscopy.

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1953 Jan. 1 to Jan. 23,'54	1953 Nov. 29 to Dec. 26,'53	1952 Jan. 1 to Jan. 24,'53	1952 Nov. 30 to Dec. 27,'52
Anterior Poliomyelitis	6	63	6	29
Chickenpox	192	189	176	272
Diphtheria	0	0	3	0
Diarrhoea and Enteritis, under 1 yr.	8	19	1	21
Diphtheria Carriers	0	0	0	0
Dysentery—Amoebic	0	0	0	0
Dysentery—Bacillary	0	5	0	4
Erysipelas	1	3	0	2
Encephalitis	0	2	0	1
Influenza	4	12	1	15
Measles	58	63	539	615
Measles—German	0	0	5	2
Meningococcal Meningitis	0	3	4	3
Mumps	62	58	127	134
Ophthalmia Neonatorum	0	0	0	0
Puerperal Fever	0	0	0	0
Scarlet Fever	61	65	40	61
Septic Sore Throat	5	6	0	2
Smallpox	0	0	0	0
Tetanus	0	0	0	0
Trachoma	0	0	0	9
Tuberculosis	7	63	12	63
Typhoid Fever	0	0	0	0
Typhoid Paratyphoid	0	0	0	0
Typhoid Carriers	0	0	0	0
Undulant Fever	0	1	0	1
Whooping Cough	4	18	8	29
Gonorrhoea	101	141	110	93
Syphilis	3	12	7	9
Infectious Jaundice	14	18	12	15

Four-Week Period January 1st to January 23rd, 1954

DISEASES (White Cases Only) *Approximate population.	*809,000 Manitoba	*861,000 Saskatchewan	*3,825,000 Ontario	2,952,000 Minnesota
Anterior Poliomyelitis	6	2	....	6
Chickenpox	192	338	2105	....
Diarrhoea & Enteritis under 1 year	8	18	....	....
Diphtheria	....	....	....	2
Diphtheria Carriers	....	....	....	....
Dysentery—Amoebic	....	....	....	1
Dysentery—Bacillary	....	1	26	10
Encephalitis Epidemica	....	1	1	....
Erysipelas	1	....	5	....
Influenza	4	....	8	3
Infectious Jaundice	14	35	113	159
Measles	58	86	737	30
German Measles	....	3	46	....
Meningitis Meningococcus	....	....	8	6
Mumps	62	265	776	....
Ophthal. Neonat.	....	....	....	....
Puerperal Fever	....	....	....	....
Scarlet Fever	61	31	413	117
Septic Sore Throat	5	13	5	17
Smallpox	....	....	....	....
Tetanus	....	....	....	....
Trachoma	....	....	....	....
Tuberculosis	7	22	88	6
Tularemia	....	....	....	....
Typhoid Fever	....	2	1	....
Typh. Para-Typhoid	....	....	....	....
Typhoid Carriers	....	....	....	....
Undulant Fever	....	....	2	7
Whooping Cough	4	4	137	17
Gonorrhoea	101	....	160	....
Syphilis	3	....	60	....

Deaths from Reportable Diseases, January, 1954

Urban—Cancer, 62; Pneumonia, Lobar, 2; Pneumonia (other forms), 12; Poliomyelitis, 1; Tuberculosis, 1; Diarrhoea and Enteritis, 2; Streptococcal Sore Throat, 1; Benign Neoplasm, 1. Other deaths under 1 year, 23. Other deaths over 1 year, 242. Stillbirths, 16. Total, 281.

Rural—Cancer, 37; Influenza, 2; Lethargic Encephalitis, 1; Pneumonia, Lobar, 6; Pneumonia (other forms), 8; Poliomyelitis, 1; Tuberculosis, 2; Diarrhoea and Enteritis, 3; Meningococcal Infections, 3. Other deaths under 1 year, 16. Other deaths over 1 year, 200. Stillbirths, 10. Total, 226.

Indians — Cancer, 1; Pneumonia (other forms), 6; Tuberculosis, 1; Diarrhoea and Enteritis, 1. Other deaths under 1 year, 1. Other deaths over 1 year, 5. Stillbirths, 1. Total, 7.

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Poliomyelitis—Reports are not all in yet but the final total for 1953 will be in the neighborhood of 2,400 cases and (at present) 88 deaths. This disease is carrying over into the new year much as it did in 1953 and this points out the fact that the virus is still present with us and not all people are immune. The type of virus isolated in all cases tested was Brunhild.

♦

At this time I wish to express my appreciation to the physicians in Manitoba who have been so co-operative in reporting cases, completing questionnaires, giving gamma globulin, etc. No doubt many times you must have thought that civil servants love paper work! I can assure you that we do not like it any more than you do but thousands of reports when carefully checked may produce some valuable information and anything we can do to conquer poliomyelitis is worthwhile! We in turn have tried to give prompt service sending out gamma globulin, information, etc., so don't hesitate to call upon us and we will not forget you!